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AMERICAN  
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ASSOCIATION

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TWENTY-FIFTH ANNUAL MEETING  
OF THE  
AMERICAN  
GASTRO-ENTEROLOGICAL  
ASSOCIATION

HELD AT THE  
RACQUET CLUB, WASHINGTON, D. C.  
May 1st and 2nd, 1922

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## CONTENTS

|  |      |
|--|------|
| List of Officers.....  | iv   |
| List of Members.....   | v    |
| List of Deceased Members.....  | viii |
| Constitution and By-Laws.....  | ix   |
| The Relations of Functional and Organic Disease of the Stomach (Presidential Address). Allen A. Jones.....   | 1    |
| Sausage Skin as an Aid in Esophageal Diagnosis. Armistead C. Crump.....  | 6    |
| The Clinical Importance of the Chronic Changes in the Appendix which are Discovered by the Roentgen Ray. Franklin W. White.....                              | 17   |
| A Preliminary Report on the Treatment of Carcinoma of the Esophagus with Colloidal Selenium. Elmer B. Freeman.....   | 35   |
| The Rôle of Spasticity in Diseases of the Digestive Tract (Case of Visceral Tetany, Causing Acute Cholangitis and Pangreatitis). Jacob Kaufmann.....         | 49   |
| Peptic Ulcer with Deformities of the Viscus, Evidenced by X-Rays, Changed for the Better by Treatment. Max Einhorn.....                                      | 66   |
| Veils in the Right Hypochondrium. Lewis G. Cole.....   | 86   |
| The Colon. Martin E. Rehfuess.....   | 90   |
| Some Underlying Causes in the Pathology of the Colon. Jerome M. Lynch.....   | 106  |
| The Early Diagnosis of Carcinoma of the Upper Colon. Milton M. Portis and Sidney A. Portis.....  | 111  |
| Treatment of Spastic Constipation. Charles D. Aaron.....   | 118  |
| Carcinoma of the Pancreas: A Clinical Study of 138 Cases. George B. Eusterman.....   | 126  |
| Studies of Gastric Secretion and Occult Blood. A. H. Aaron, E. C. Beck and H. C. Schneider.....  | 138  |
| H-Ion Concentration Method of Gastric Analysis. John H. King and Samuel Halpern.....   | 151  |
| Local Treatment of Gastric Irritation. Charles G. Stockton.....  | 163  |
| The Gastric Disturbances of Old Age. Julius Friedenwald and Theodore H. Morrison.....  | 171  |
| The Diagnosis of Intestinal Obstruction. Alexius McGlannan.....  | 184  |
| Intestinal Infection with Entamoeba Histolytica as a Factor in Arthritis Deformans. Sidney K. Simon.....   | 196  |
| Intestinal Toxemia: Enterotoxines Allied to Adrenalin in Composition which Cause Rise in Blood Pressure and Increased Sympathicotonus. John C. Hemmeter..... | 201  |
| The Diagnostic and Therapeutic Value of Transduodenobiliary Drainage. E. L. Eggleston.....   | 206  |
| Visceral Adhesions and Bands: Normal Incidence (Second Paper). John Bryant.....  | 223  |

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# CONSTITUTION AND BY-LAWS OF THE AMERICAN GASTRO-ENTEROLOGICAL ASSOCIATION

## CONSTITUTION

**ARTICLE 1.** This Association shall be known as the American Gastro-Enterological Association.

**ARTICLE 2.** The object of this Association shall be the promotion of investigation of the normal and pathological conditions of the digestive organs.

**ARTICLE 3.** Membership in this Association shall be of five kinds: (a) Honorary, (b) Corresponding, (c) Active, (d) Associate, (e) Emeritus.

(a) Honorary membership shall be restricted to such as have attained an international reputation by their published works and have scientifically furthered the subject of gastro-enterology. Their nomination shall take place at the annual meeting, upon the written suggestion of at least six active members and on the recommendation of the Council of the Association at the next annual meeting. Their election shall be by unanimous vote of the members present.

(b) Corresponding membership shall be restricted to foreign scientists, in recognition of meritorious work within the scope of the Association. Candidates for such membership shall be proposed by at least six active members and receive the recommendation of the Council at the next annual meeting. Their election shall be by unanimous vote of the members present.

(c) Active membership shall be restricted to American and Canadian investigators and practitioners who have published meritorious work in normal or pathological anatomy or physiology, in medicine or surgery of the digestive canal and its secretory appendages, and who enjoy an unimpeachable moral standing in the medical profession.

Names of candidates for active membership must be: (1) Proposed by two active members who are not members of the Council, such proposal to be submitted to the Committee on Admissions and Ethics, which should report to the Council as soon as possible; (2) must be recommended by the Council and (3) must receive a majority of the votes of active members present at the annual meeting of the Association.

(d) Associate membership shall be restricted to American and Canadian investigators possessing the general qualifications requisite for active membership, and the method of election shall be identical. The number of such Associate members shall not exceed twenty-five. They shall enjoy all the privileges of the Association with the exception of attending the Executive Sessions at the annual meetings. Such Associate members shall constitute a list from which Active members may be chosen, but if at the expiration of five years an Associate member has not been elected to active membership, he shall then be automatically dropped.

(e) Emeritus membership shall be restricted to those previously active members whom the Association thus elects to honor. Candidates for such membership shall be nominated by the Council. Their election shall be by a majority vote of

the Active members present at the annual meeting of the Association. Such members may enjoy all of the privileges of the Association, except that they may neither vote nor present papers at the annual meetings.

The election of members shall take place at the annual meeting of the Association and shall be by ballot.

It shall be the duty of the Secretary to notify Active members of all nominations that are to be submitted to the Association, not less than one month previous to the date of the annual meeting.

ARTICLE 4. The officers of the Association shall be a President, two Vice-Presidents, a Secretary, a Treasurer, and a Recorder; also, three specially chosen active members to be known as Councilors, who, together with the officers, shall constitute the Council of the Association. The said Councilors shall be elected as follows: one for one year, one for two years, and one for three years. At each subsequent annual meeting, one member shall be elected for the term of three years to fill the annual vacancy.

ARTICLE 5. There shall be a Committee on Admissions and Ethics. It shall consist of the President, the Secretary, and three other members not members of the Council. These members shall be elected at the annual meeting at which this amendment shall be adopted for the following terms: one for three years, one for two years and one for one year. At each subsequent annual meeting one member shall be elected for the term of three years to fill the annual vacancy. No member of the Committee shall be eligible for re-election until after one year shall have elapsed from the end of his term of service. It shall be the duties of the Committee on Admissions and Ethics to examine the merits of a candidate, to investigate any charges made against a member, and to report the results of the examinations and investigations to the Council in writing in the shortest possible time. It shall also be the duty of this Committee to study the active work in science and practice of gastro-enterology accomplished in this country and Canada, and to invite the meritorious workers to join this Association.

ARTICLE 6. The election of officers, Councilors and members of the Committee on Admissions and Ethics shall take place at the annual meeting and shall be by ballot.

ARTICLE 7. Vacancies in the offices occurring in the interval between the annual meetings shall be filled temporarily by the Council.

ARTICLE 8. The annual meeting of the Association shall be held at a time and place to be decided by the Council.

ARTICLE 9. The President shall be the chairman of the Council.

ARTICLE 10. There shall be annually two meetings of the Council: one shall take place at the call of the President, shortly after the annual meeting of the Association; the other shall take place at least four weeks previous to the annual meeting. It shall be within the discretion of the President to conduct this latter meeting by correspondence.

ARTICLE 11. The Council shall manage the affairs of the Association in accordance with the Constitution and By-Laws, and its minutes shall be reported to the Association at the annual meeting.

ARTICLE 12. This Constitution may be amended by a two-thirds vote of all the active members at any annual meeting, provided that notice of proposed amendments has been given in writing to all the active members of the Association by the Secretary, and provided further that such proposed amendments shall have been submitted to the Council and have been held over one year.

ARTICLE 13. Any member failing to attend three consecutive annual meetings without an excuse acceptable to the Council shall be dropped from the roll. The Secretary, however, shall be required to call the attention of a delinquent member to the facts in his case previous to the third meeting.

ARTICLE 14. This Association adopts the Code of Ethics of the American Medical Association. Charges of advertising, of publishing knowingly false scientific statements, of actions unbecoming a gentleman and a high-standing physician, etc., shall be investigated by the Committee on Admissions and Ethics. The accused shall have liberal opportunities to clear himself of the accusation. The Committee shall report its findings to the Council, and on the recommendation of this body an offending member may be expelled by a three-fourths vote of those present at the annual meeting.

### BY-LAWS

ARTICLE 1. The President and Vice-Presidents shall discharge such duties as are implied by their respective offices. The President shall preside at all sessions of both Council and Association.

ARTICLE 2. The Secretary shall attend to the usual clerical duties of the Council and the Association.

He shall keep on file all applications for membership for a period not to exceed ten years, together with all data pertaining thereto. On retirement of a secretary, all such data shall be transmitted to his successor.

ARTICLE 3. The Treasurer shall collect and keep a record of all payments and arrearages of dues, and shall report on both at the annual meeting. He shall keep all other accounts of the Association, shall make payments from the Treasury upon order from the Secretary or other adequate authority, and shall render a statement at each annual meeting. His accounts shall be audited annually by a committee appointed by the President.

ARTICLE 4. The Recorder shall secure all papers read at the annual meeting, prepare corrected discussions thereof, and supervise the publication of the annual volume of the Transactions.

ARTICLE 5. The Council shall report its transactions to the annual meeting of the Association. It shall superintend the publication of scientific papers, but shall not appropriate for such printing an amount exceeding the amount of \$100, without the vote of the Association. The order of business shall be arranged by the Council before the annual meeting. It shall pass upon the eligibility of candidates for membership.

ARTICLE 6. The entrance fee of Active members shall be ten dollars.

The annual dues of Active members shall be fifteen dollars, payable in advance.

The annual dues of Associate members shall be ten dollars; such members shall not be required to pay an entrance fee when finally admitted to active membership.

Laboratory workers, Honorary, Corresponding, and Emeritus members shall be exempt from the payment of dues.

It shall be the duty of the Treasurer to report all members who are in arrears, to the Council. Any member whose dues shall be in arrears more than two years, shall be reminded of the fact by the Treasurer, in writing; in event that payment should not then be made, he may, on vote of the Council be dropped from the roll of the Association.

ARTICLE 7. When sufficient money is in the treasury, the membership dues may be omitted when decided by the Council.

ARTICLE 8. These By-Laws may be amended, repealed or suspended by a two-thirds vote of the members present at any meeting of the Association.

ARTICLE 9. The quorum of the Association for the transaction of business, but not for the reading of papers, shall consist of seven active members.

ARTICLE 10. All titles of papers submitted for reading at the annual meeting shall be sent to the Secretary not later than four weeks before the date of the meeting.

ARTICLE 11. A typewritten abstract of not over three hundred words, of every paper to be read, must be sent to the Secretary at least four weeks before the annual meeting. It shall be the duty of the Secretary to send to each member of the Association a complete list of the papers to be read at the annual meeting, with their full titles, at least two weeks before the annual meeting.

ARTICLE 12. Papers submitted to be read before the Association shall not have been previously published or read elsewhere. Papers shall not exceed the limit of fifteen minutes, without the consent of the majority of members attending.

ARTICLE 13. Members shall be required to leave their manuscripts with the Secretary or the Recorder, before the close of the annual meeting.

ARTICLE 14. The Recorder shall require the return of proof within two weeks, otherwise manuscript shall be published without revision by its author.

# THE RELATIONS OF FUNCTIONAL AND ORGANIC DISEASE OF THE STOMACH

## PRESIDENTIAL ADDRESS

ALLEN A. JONES

*University of Buffalo, Buffalo, New York*

Some people are susceptible to functional gastric disorders from exciting factors which in others would pass unfelt. It is a common observation that one person will choke and retch at the application of a tongue depressor while another placidly allows the examination with no manifestations of neuromuscular disturbance. The natural or acquired nervous control of some patients is apparent as every step of a physical examination is passed through; while others present neuro-psychic, neuromuscular, or neurocirculatory upsets. We may note an almost endless variety of reactions in different patients to our questions and examinations. This is a practical point which may well be noted and borne in mind as we pursue our clinical studies. The stomach in a nervous individual is apt to react excessively to an organic irritation and this hyperesthesia is prone to exaggerate the symptoms of disease but, on the other hand, may serve to attract attention to a lesion in its mild or more easily curable stage, as for instance a gastritis or a small, superficial ulcer.

It is the disturbance of function which in a large measure produces the symptoms of an organic disease. One may readily recall, however, the discovery of quite advanced disease in some cases wherein the functions of the stomach went stolidly and placidly on in spite of the organic changes. These experiences put us on guard in the investigation of cases and the detection of disease.

As psychic, emotional, and reflex influences induce disorders of the secretory, motor or sensory innervation of the stomach so organic disease may be similarly influential.

In most ulcer cases gastric secretion is apparently overactive. The preulcerative stage is probably one of high acidity, irregular peristalsis and pyloric spasm which persists and is exaggerated after the initiation of ulcerative tissue damage. This applies as well, and perhaps

more emphatically, to duodenal ulcer. The sensory response in the event of actual ulceration usually assumes prominence and thus all three, secretory, motor and sensory disturbances, henceforth play important rôles in the symptom complex. Efforts to relieve the patient with either gastric or duodenal ulcer are successful in proportion as we are able to control the acidity, the spasm and the pain, and this control of the disturbed functions goes far toward the cure of the ulcer.

Not only is pylorospasm an ordinary attendant of gastric ulcer but spastic behavior is noted in other parts of the organ and peristaltic unrest is a common feature. Hypertrophy of the gastric wall may result and its irritability become so marked that an aggravated erethism is produced. With ulcer near the pylorus this is manifested clinically by curtailment of the period of relief following the ingestion of food in chronic recurring cases. Fatigue of the motor function follows in some instances and indeed, distinct atony may be observed. The secretory function also shows variations and low acid values may be present in some stages of the ulcer syndrome.

Pain is also an uncertain and fickle quantity. It is not a reliable index of the extent or severity of the ulcerative process; it is often responsive to nervous perturbation or fatigue and under some circumstances is most annoying when the patient is on an approved diet. When these vagaries occur during the ambulatory treatment of an ulcer case the relief attending physical, mental and emotional rest emphasizes the important rôle played by the nervous mechanism. Indeed, the treatment of either gastric or duodenal ulcer at some stage in its course demands real and effective general rest in order that the stomach may experience the rest its more normal functioning affords. Thus the ambulatory treatment of ulcer is not a proper criterion upon which to base results. Complete success often follows when effective rest is established.

Few organs in the body reflect nervous and physical fatigue so readily as does the stomach. Vomiting has long been classed as a motor neurosis and it is an extremely variable factor in organic disease. As compared with pylorospasm and pain it is uncommon in gastric and duodenal ulcer, is perhaps more often present than these in gastritis while in cancer it is apt to occur frequently. With persistent pyloric spasm in ulcer or as a result of cicatricial stenosis vomiting is prone to supervene. Profuse gastrorrhagia in ulcer induces hematemesis. Many cases of ulcer, however, are observed in which vomiting is pres-

ent without undue retention of gastric contents and without hemorrhage, and in these it seems an expression of hyperesthesia.

With gastrectasis, gastroptosis, gastritis, gastro-duodenitis, enteritis, entero-colitis or intestinal obstruction vomiting more or less commonly occurs but it is a symptom so frequently present with disease elsewhere than in the digestive tract itself that it is not diagnostic unless it has special characteristics.

Even in the presence of serious gastrointestinal disease the tolerance of the stomach may be quite wonderful or its intolerance be most disquieting. We have probably all seen cases with retention of gastric contents for several days yet but little, if any, vomiting and on the contrary cases with less retention in which vomiting occurred two or three times a day. It is difficult to explain these differences in the behavior of the stomach but they are doubtless linked up with some imbalance in the autonomic nervous system. It would seem that many cases of the ulcerative and preulcerative state are associated with vagotonia whether or not infection is a determining factor in the actual lesion. Perhaps there are some other influences at play in the gastrointestinal tract which may furnish a partial explanation of the phenomena.

Keith's nodes, special activating sensitive areas in the gastrointestinal tract, may exert a large influence in diseased conditions. Recently Walter C. Alvarez (1) with his specially devised electrogastrograph has found that there is apparently a gastric pacemaker situated "on the lesser curvature near the cardia and his electrogastrograms show that definite electrical disturbances are travelling over the fundus when no movement of the muscle can be detected even with a magnifying glass." He found, however, that "there are shifts of the pacemaker sometimes even to the pyloric region; there are contractions appearing here and there which do not seem to set up any travelling disturbances; there are waves which travel half way across the stomach only to be blocked suddenly at some point; and there are at times systoles of the stomach as a whole." He further found "that the body of the stomach and the pyloric antrum can contract with separate rhythms." In studying the behavior of the duodenum Alvarez observed that "the approach of a wave in the pars pylorica is the signal for the appearance of a strong tonus wave in the first portion of the duodenum. If this can be shown also in man, we shall take a step forward in explaining the production of pain in duodenal ulcer." Alvarez further observed that peristaltic waves passing down the intestine had their

origin in gastric waves and he mentions the work of Reynolds and McClure (2) in their fluoroscopic study of the motor phenomena in normal stomachs and in the presence of peptic ulcer and its pain. They observed modifications of gastric peristalsis in fourteen out of sixteen ulcer patients studied. "The abnormalities noted were: (a) An exaggerated type of normal gastric peristalsis; (b) irregularity in the time of occurrence, depth and length of the course of peristaltic waves; (c) partial or complete intermittent spasm of the pyloric sphincter; (d) localized, permanent, stationary spasm of the gastric musculature causing the so-called incisura; (e) gastric antiperistalsis; (f) delayed emptying time of the stomach; and (g) very rapid emptying of the stomach. They mention the work of Cannon, Cole, Carlson, Hardt, Hamburger, Tumpowsky and Ginsburg, Homans, Luckhardt, Phillips and Hurst.

While one or more of these motor disturbances occurred with the onset of pain and disappeared with its subsidence some cases presented them in the absence of pain and other cases had pain without abnormal motor manifestations. The association of these peristaltic changes with pain does not prove that they are the cause of the pain but it was noted that giving alkalis relieved the pain and normalized the peristalsis. While there are probably other factors which are operative in the production of pain in ulcer cases it is noteworthy that alkalis relieve the pain more effectively in most cases, than any other remedies we employ; and one cannot escape the clinical conviction that the acidity is an influential factor in the production of both the pain and the motor phenomena.

Let me remind my hearers that Max Einhorn devised a gastroph, many years ago, with which to record something of the motor activity of the stomach.

In reference to the chief functional disturbances going with gastritis it may be observed that in the acute form they correspond closely to the physiologic law that an acutely inflamed organ demands rest—therefore vomiting, distress, eructation, etc., follow the ingestion of any but the most soothing foods such as milk in suitable form. In cases of chronic gastritis in which the morbid changes impair the glandular and muscular structures diminished secretion and motion naturally result. Strictly speaking it is impossible for a stomach which is the seat of chronic inflammation, or even chronic passive congestion, to functionate perfectly, but many cases are seen with apparently minor functional disturbances while other cases daily present rather stormy

symptoms, an explanation of which is often found in a neuropathic state.

In achylia gastrica, which may be the result of glandular atrophy, the writer has more frequently observed eructation, precipitate emptying, etc., in neurotic or nervously fatigued individuals than in others. The diarrhea of achylia gastrica seems, in a measure, to be a nervous manifestation in some cases wherein this disorder is observed. A few cases of achylia in my experience have been associated with cholelithiasis and a few with syphilis. Indeed the functional disturbances of organic gastric disease may be associated with and responsive to organic disease elsewhere.

As this short address is merely to open or introduce your more deliberate and scientific discussions I will close with a few words upon the functional difficulties of marked gastro-enteroptosis. We seldom see patients with this disorder in a state of normal health. They tire quickly and recuperate slowly; they are underweight and are apt to present the manifestations of neurocirculatory asthenia. While the stomach may empty itself on time there is usually colonic stasis. Gastric atony and a pathologic residue are often seen, belching is common, vomiting rare, unless induced by some intercurrent or complicating disorder, appetite is apt to be fitful, the tongue is frequently coated, the condition is often present with a mixed up complex of neurasthenic symptoms. If a few pounds of weight are gained during sanitarium or rest treatment they are promptly lost on resumption of life's activities and duties. There seems to be something in the bodily habitus, the digestive organs and the endocrine, nutritional and metabolic conditions of these patients preventing them from attaining vigorous health. Perhaps the something dates back to their ancestry, perhaps hereditary tendencies are aggravated by early faulty environment as to physical education and habits. Possibly the drag on the mesentery, the static splanchnic circulation, the pressure of the superior mesentery artery and vein crossing the duodenum may have considerable influence. At all events experience has shown me that efficient lower abdomen support is one of the most potent therapeutic measures aiming toward the amelioration of symptoms and a moderate gain in weight.

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## SAUSAGE SKIN AS AN AID IN ESOPHAGEAL DIAGNOSIS

ARMISTEAD C. CRUMP

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I deem it a great honor to be called upon to present to you my ideas regarding the use of the sausage skin as an aid in diagnosis of diseases of the esophagus. It is to be strictly understood, as the title shows it to be, only an aid along with the other methods commonly used in this difficult part of the gastrointestinal tract. As a rule it is the simplest thing to have a routine of examination in these cases. First, naturally a bougie examination with a flexible staff and large olive about 35-F.—small olives dangerous. If done with care and without using any but the gentlest force it should not be any more dangerous than passing the usual stomach tube. By this means we are able to find out the location accurately of the point of obstruction. If an obstruction is found it is best to order the usual X-ray examination, which means fluoroscopic and plates by the thick bismuth paste method, as advocated by Hirsch, and try to get some idea as to the appearance the obstruction will give; whether the change is characteristic of carcinoma, diverticula or cardiospasm. This unfortunately cannot tell the extent of the involvement as not enough bismuth would be passed down from a bolus of paste mixture to fill out properly the esophagus and give an idea of the lower reaches of any obstructive process. This is important if radium treatment is to be used in epithelioma. It will serve also to confirm the diagnosis of cardiospasm or diverticula.

Of course, the idea of painting over the esophagus would be a good one if it were possible, but the peristaltic waves rapidly take the bismuth to the stomach at a rate of speed varying according to the amount of obstruction. There are certain very important things, however, to be found out by giving bismuth by mouth which are characteristic of neoplasm, namely, cupping, pockets, or a moth eaten appearance. In the "sausage skin" method this can be accomplished where necessary by selecting a leaky skin. In an early condition where ulceration has not occurred and there is no evidence

other than a hardened indurated esophageal wall—X-ray, esophagoscope and even a specimen may fail to reveal the true pathology, as I will show later. So the best we can do is to eliminate as far as possible the other disease and realize that 80 to 90 per cent of such invasion (1) in the esophagus is of malignant nature and even with a tubercular or specific condition you can have an implanted neoplasm.

The esophagoscope is extremely valuable and where possible should always be used to get the appearance of any growth as a possibility of making a diagnosis by this means. However, the surgeons frequently object to a specimen being removed as claim is made that it is likely to hurry the process of growth by stimulation and also likely to cause metastasis. When done it should be just previous to operation.

#### USE OF SAUSAGE SKIN

First of all it must be remembered that we are really introducing a bag which is limited in diameter to a size about double that of the esophagus, and naturally may be said to veil somewhat the surface irregularities similar to a veil covering an ulcerated tumor of the breast. This can be overcome, as before noted, by either selecting a leaky skin or making a puncture at a measured point on the bougie that will be proximal to the obstruction in order to allow the bismuth mixture to leak out. The selection, preparation and use of the sausage skin was described by me some years ago in the *Journal of the American Medical Association* (2) and later in *Johnston's Operative Therapeutics* (3). Stewart (4) has used and continues to use this method with success. Skins ready for use, properly cured, selected and preserved in salt can now be obtained from a well known slaughter house.<sup>1</sup> The use of the plain skin has been very satisfactory, but frequently it takes quite a time to get the patient "instrument-trained" before making a satisfactory plate. To facilitate the quick placing and withdrawal of the skin a special bougie has been devised after considerable experimenting. Of course, indiscriminate bougieing is a dangerous practice and should only be done with the greatest caution. Forceful means should never be resorted to and only the gentlest manipulation of instruments. A guide should always be used when it is desired to pass an instrument by a stricture, even when working through an esophagoscope. One can either use the "silk thread" method advocated by Plummer (5) or

<sup>1</sup> Manager Swift & Co., Harrison Branch, Kearny, N. J.

the "flexible-tipped" piano wire which I shall later describe. If the preliminary X-ray examination shows either a cardiospasm or diverticula the "silk thread" method should only be used. However, in cardiospasm the "sausage skin" method is not indicated.

#### INSTRUMENTS

##### *Description of sausage skin bougie (figure 1)<sup>2</sup>*

A. Proximal portion flexible metal tube no. 28-F. in size and 12 inches in length. At its center is an aluminum hub with a shallow groove for tying proximal end of sausage skin, also a deep groove for patient to grasp between the incisor teeth, deep groove being exactly 6 inches from distal end. At distal end (*J*) is a metal tip having three large radial openings to facilitate rapid filling and emptying of skin. Axially also there is a threaded opening for attachment of bougie (*B*). At the opposite or proximal end is a tip for attaching a ten or twelve ounce rubber bulb.

B. Flexible bougie composed of sections of no. 18-F. Silk-catheter tied to metal connections (*I*) made of Monel-metal. These metal tubes have at their centers lateral openings for aiding in filling and emptying the sausage skin cover (fig. 3). They are 1 inch in length and placed 2 inches apart—center to center. The long portion is 10 inches in length—this can be extended 2 inches by adding section "*K*," "*L*," is the tip which is threaded for attachment to bougie and grooved for tying distal end of sausage skin. Axially is a small hole so that the instrument can be guided in place over a silk thread or piano wire (no. 32 English gauge).

C. "Primer" for depositing a thick bismuth mixture outside of sausage skin used in place of puncturing skin when stricture is in upper one-third.

Figure 3 shows instrument assembled—covered with sausage skin which is tied at the hub (6 inches), attachment of bougie and tip. The instrument is inflated with the bismuth mixture:

R̄—Muc. Acacia  
Bis. Subcarb aa   ℥ viii  
Aqua rad. O T

by pressing bulb. Bulb should have a capacity of about ℥ xii.

<sup>2</sup> Geo. Tiemann & Co., 107, East 28th St., New York will be prepared to furnish instruments.

*Description of guide wire and carrier (figure 1)*

"F." "Flexible tipped guide wire" made of no. 19 English gauge piano wire 20 inches long. Attached to distal end is a small olive no. 7-F. which joins the wire to a section of "eye glass" cable  $2\frac{1}{2}$

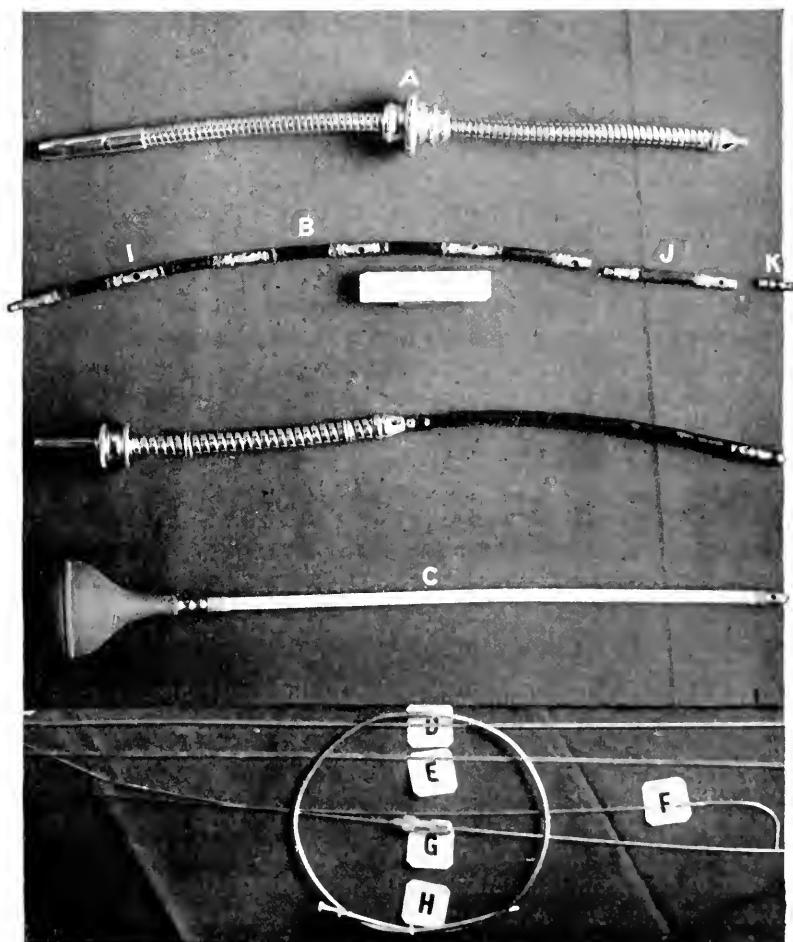


FIG. 1

inches long. The tip of the cable having a round ball  $2\frac{1}{3}$  mm. in diameter. Illustration shows flexibility of tip.

"G." "Chuck" which when tightened up is used to twirl flexible tipped piano wire.

"H." "Carrier" Flexible metal tube  $3\frac{1}{3}$  mm. outside diameter made of no. 27 English gauge Monel metal  $3\frac{2}{3}$  mm. broad wound spirally to form a flexible tube. This is made in two sections of 9 inches each which are joined in the center by a screw connection.

"D." "Obturator" which is only used to preserve the shape of "carrier."

*Technique of passing "guide wire" (figure 2)*

- (1). Flexible tipped wire in carrier is passed to site of obstruction.
- (2). "Carrier" withdrawn 2 inches releasing flexible tipped wire.
- (3). Chuck attached to mouth end of wire. Flexible tip is then twirled forward with a corkscrew-like motion using only enough force to shift the weight of instrument.
- (4). "Carrier" is then passed on flexible-tipped wire as a guide.
- (5). Withdraw flexible tipped guide and introduce piano wire for guiding sausage bougie. Piano wire 22 English gauge, 48 inches in length (double length of instrument).
- (6). Wire in place for directing tip of instrument in right direction.

TECHNIQUE OF X-RAY EXAMINATION WITH SAUSAGE SKIN BOUGIE

*Preliminary.* Patient presents himself for examination preferably on an empty stomach. It is not necessary to cocaine the pharynx. In some cases this may help. If the patient is in the hospital it is best to order morphine, 0.25 grain, with atropine, 0.01 grain, just before the examination.

*Passing bougie.* The instrument is very flexible and the sausage skin being slimy naturally aids in its passage. If the stricture is one of large caliber without pockets on its upper surface there is no occasion for placing the guide wire. However, if there is the least resistance met with, a guide should be used as directed above. It is preferable to pass the instrument and inflate the skin several times with plain water in order to accustom the patient to the presence of the instrument.

*X-ray examination.* After passing instrument the patient is placed first in the right oblique position—groove on hub A, (fig. 1) is then adjusted between middle incisor teeth. Rubber bulb filled to full capacity with bismuth acacia mixture is then attached. By squeezing bulb about 100 cc. is then run in slowly. The large end of bulb is then held above patient, pressure released on bulb, withdrawing any air that might be in the instrument. The bulb is again pressed until

you have filled the sausage skin to capacity, gentle pressure on the bulb being continued while plate is being made. If retching or coughing commences pressure is removed from bulb which will quickly empty the skin. After making the plate pressure in the skin is relieved by withdrawing about half of the bismuth mixture. Patient is then placed in the prone position face down. Skin is then refilled as before. During the time of exposure patient is told to "hold your breath" this being the signal that the sausage bougie is properly adjusted and the radiographer can make his exposure. Patient should not be told to take a "deep breath" as is frequently done.

*Case 1.* R. R., baby boy of two years. Referred to Dr. Torek of the Lenox Hill Hospital.

Thirteen months before examination swallowed concentrated lye. At the time patient was first seen he could swallow milk only and that with the greatest difficulty. Flexible-tipped piano wire was passed without the least trouble. He was then dilating from 5-F. to 17-F., plate (fig. 4) was then made. He was then bougied up to 27-F. Treatment had to be discontinued as patient contracted measles. This was followed by double mastoid and pneumonia. Fortunately he could take his nourishment. Treatment was recommenced and in four months from the first treatment patient was dilated to 34-F. He could eat anything. Figure 5 shows the location of the main points of stricture at 5 and 7 inches from upper incisor teeth.

*Case 2.* S. B., woman, age fifty-one, married thirty-two years.

*Family history.* Negative.

*Previous illness.* Twelve years ago gall stones with jaundice. Six years ago appendix removed.

*Present illness.* For two years coughing spells at times raising blood. Recently notices she cannot swallow solid food. When she does so she vomits it. Gastro-intestinal X-rays negative. Bougie examination shows an obstruction 11 inches from incisor teeth admitting no. 18 French olive. Sausage skin X-ray using plain skin shows annular stricture band. Patient was dilated to 40-F. She had lost 20 pounds in weight. The following year she gained all of her lost weight and added 20 pounds more. The X-ray plate shows an old tubercular process. Tubercle bacilli, however, were never found in her sputum.

*Case 3.* See figure 7. Wm. F., aged seventy-one. Occupation, bricklayer.

*Chief complaint.* Sharp stabbing pain in epigastrium after eating for two weeks—for past year noticed food would stick for few seconds. Nausea after eating in attacks for years. Gastrointestinal X-rays reported negative. Esophageal examination with bougie shows some resistance to 34-F. olive at 16 inches.

*Test meal.* Absence of hydrochloric acid. Positive lactic and guaiac test. These findings with the irregularity shown beautifully in the X-ray plate, lead to a diagnosis of epithelioma.

*Operation.* Thoracostomy finding pleuritic adhesion pulling esophagus to nearly a right angle. You will note that the cardia is 2 inches beyond the point of obstruction.

I show this case as it clearly brings out one point. All methods of examination should be used in these cases. In this particular case

an esophagoscopic examination would have been of immense value. However, patient was cured if rather extreme measures were resorted to.

*Case 4.* Figure 8. Epithelioma—lower one-third. Alto W., aged sixty-four. Occupation, caterer.

*Chief complaint.* Epigastric pain for five months.

*Personal history.* Negative except for slight rheumatism occasionally.

*Present history.* Six months ago vomited solids. For five months sharp pains in epigastrium during night. Nausea but no vomiting. Recently pains constant and cannot swallow solids. Loss of 50 pounds in six months.

P. X. Epigastric rigidity, otherwise negative. Gastrointestinal X-rays by ordinary method show obstruction at middle of esophagus, but diagnosis was not made.

Bougie examination shows obstruction  $12\frac{1}{4}$  inches from upper incisors which allows passage of 29-F. bougie.

*Case 5.* Figures 9 and 10. Epithelioma upper one-third. John S., aged fifty-one. Occupation, tinsmith.

*Chief complaint.* Swelling over chest for two weeks. For a year or two sense of pressure in chest gradually getting worse. For one month food seems to stick in throat. Bougie shows obstruction at 8 inches from upper incisors admitting 18-F. olive.

*Case 6.* Figure 11. Early epithelioma of the esophagus. James O'B., aged 31 years, laborer.

Three months ago discharged from army. One week later sharp pain in right lateral chest after eating corn beef sandwich. This lasted about ten minutes and went away. Pain on swallowing since then, but can swallow solid food. At times regurgitates food. Has lost about 10 pounds in past three months.

Bougie examination shows obstruction at  $11\frac{1}{2}$  inches admitting no. 35 French olive. Gastrointestinal X-rays negative. Esophagoscopic examination by Dr. Kernan shows mucus membrane congested, posterior wall purplish, firm, bulging and immobile. No ulceration. Specimen removed reported negative. Specimen tissue found in eye of bougie one week previously reported epithelioma. Thoracotomy was performed by Dr. Adrian Lambert, but tumor was found attached to trachia and could not be removed.

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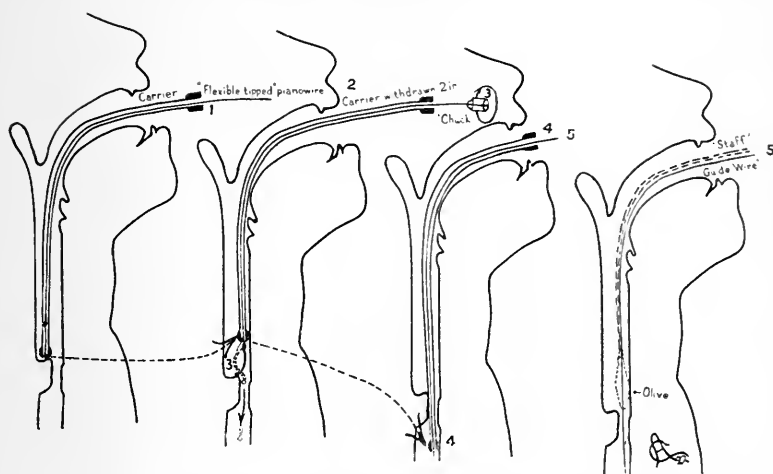


FIG. 2. SHOWING METHOD OF PLACING GUIDE WIRE FOR THE PASSAGE OF INSTRUMENTS

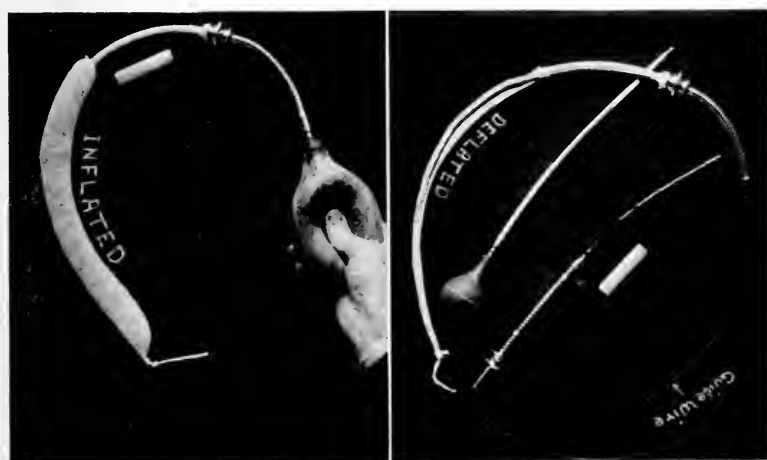


FIG. 3. "SAUSAGE SKIN" BOUGIE INFLATED WITH BISMUTH MIXTURE



FIG. 4

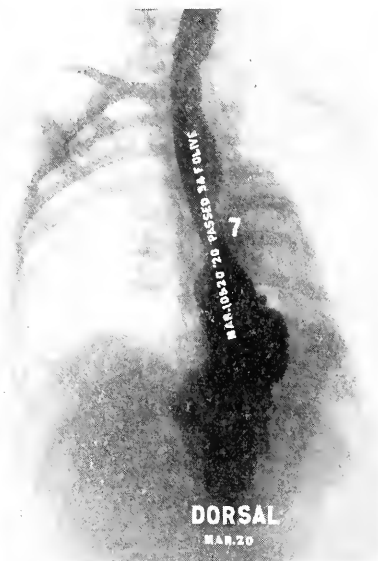


FIG. 5

FIG. 4. CICATRICAL STRICTURE FROM LYE—BABY 2 YEARS  
 FIG. 5. SAME AS FIGURE 4—FOUR MONTHS AFTER TREATMENT

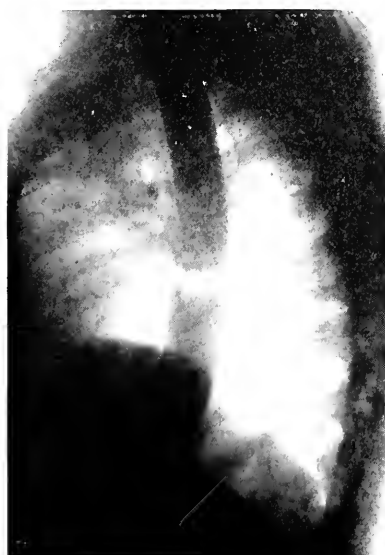


FIG. 6



FIG. 7

FIG. 6. ANNULAR STRICTURE SECONDARY TO RUPTURE OF TUBERCULAR GLAND INTO ESOPHAGUS  
 FIG. 7. NARROWED LUMEN FROM PLEURITIC ADHESION

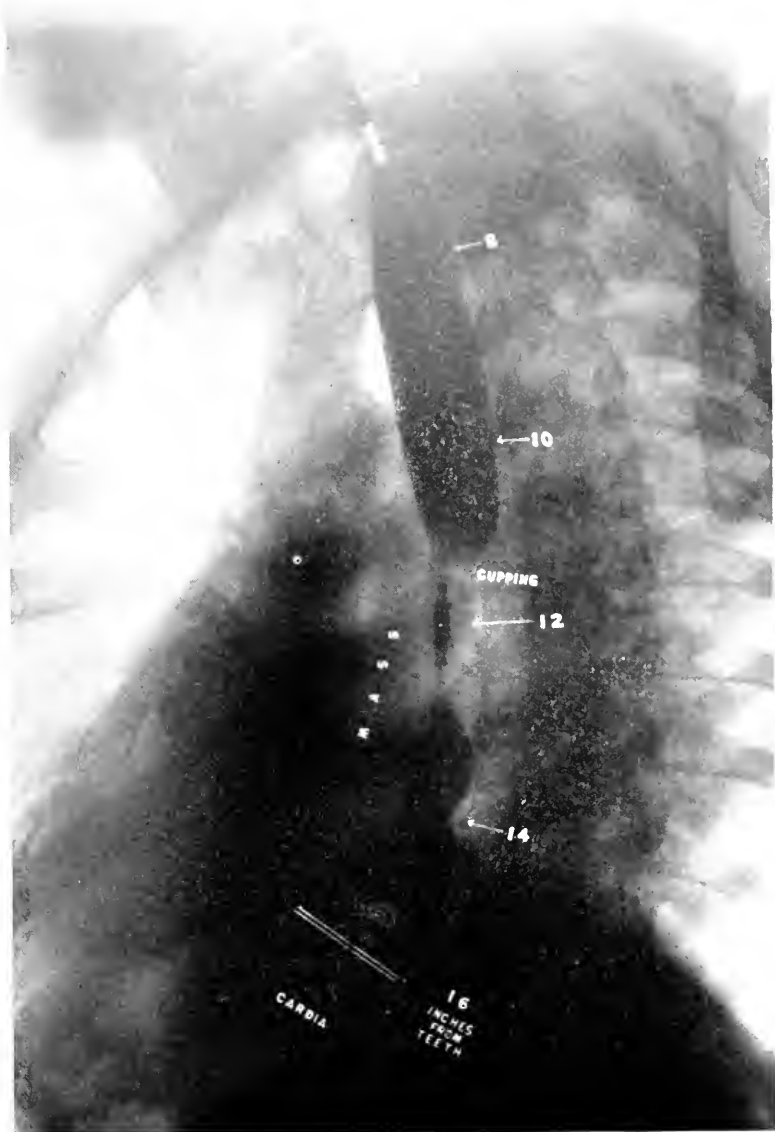


FIG. 8. EXTENSIVE EPITHELIOMA INVOLVING PART OF MIDDLE AND LOWER ONE-THIRD



FIG. 9



FIG. 10

FIG. 9. LOCALIZED EPITHELIOMA ONLY ONE INCH IN EXTENT

FIG. 10. ANTERO-POSTERIOR VIEW OF FIGURE 9



FIG. 11 (A)

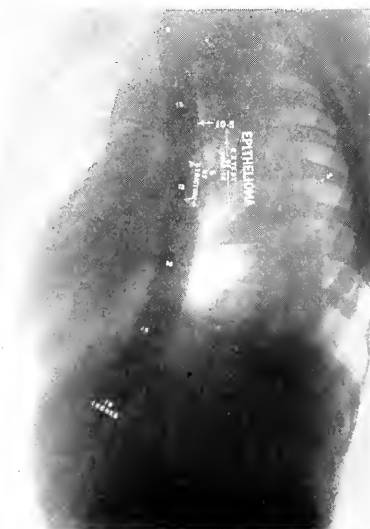


FIG. 11 (B)

FIG. 11 (A). }  
 FIG. 11 (B). } EARLY EPITHELIOMA OF ESOPHAGUS MIDDLE ONE-THIRD

## THE CLINICAL IMPORTANCE OF THE CHRONIC CHANGES IN THE APPENDIX WHICH ARE DISCOVERED BY THE ROENTGEN RAY

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"Chronic appendicitis" is a diagnosis which is very common and rather loosely applied by the clinician, the surgeon and the radiologist. The symptoms are not clean cut, the surgical findings are very variable, and the X-ray findings equally so. There is a common impression that almost every adult has some chronic changes in the appendix, and therefore we can make the diagnosis very frequently and be backed up at operation or by the pathologist. The question resolves itself into two parts: first, what are the signs of chronic changes in the appendix? Second, how important are they to the individual?

We may say that the radiologist is only concerned with the diagnosis. Let him make a record of the facts and hand them to the family doctor. Theoretically, the X-ray data must be taken with the clinical data to decide the diagnosis and treatment. Practically, the radiologist has a heavy responsibility in making the diagnosis and in talking to the family doctor about it. Consciously or unconsciously what he says carries great weight. He shows pictures, clear graphic evidence, which is apt to be overemphasized. Many times merely to mention or suggest "a chronic appendix" is equivalent to saying to the family doctor, "Take it out at once." No other organ is so frequently removed without cause. On this account we must discuss not only the first question, the diagnosis, the signs of chronic appendicitis, but also the second question, how important they are to the individual patient. In fact the second question is more important at present than the first. You are experienced men, you know the signs of chronic changes in the appendix. I have no new sign to offer. My only excuse for taking them up here is because there has been such a wide difference of opinion on details in the past. It is well to discuss them briefly and get together on essentials.

## PATHOLOGY

Chronic appendicitis is a poor term. There are practically no chronic inflammations in the appendix with the rare exception of tuberculosis. There are chronic changes, the result of recurrent previous inflammation which may cause chronic functional disturbance. In acute inflammation, we have necrosis, ulceration, which leaves scar tissue, fibrosis and peritoneal adhesions; the interstitial tissue contracts, the mucosa is destroyed, and we have obliterative changes. The connective tissue in the appendix, the slow progressive fibrosis may lead to marked changes in form without symptoms, especially in old people and the condition was formerly first discovered at operation or autopsy. Now it is frequently discovered by the X-ray. The connective tissue in adhesions is often a cause of symptoms but often is not.

## X-RAY SIGNS

Direct signs in the appendix are tenderness, fixation, kinking, changes of shape, abnormal position, lack of filling, slow emptying, beading, also adhesions in the ileocecal region. The indirect signs are pyloric spasm, gastric residues and ileal stasis. The signs themselves are quite clean cut and easily understood. Their interpretation requires exact knowledge of the anatomy and physiology of the bowel. Most of the signs are suggestive rather than definite, and several, the more the better, are needed for a diagnosis (taken of course with the clinical evidence). If there is no tenderness and no fixation, the other signs count for little. Up to a few years ago there was the greatest divergence of opinion on the significance of signs such as the filling and emptying of the appendix, ileal stasis, etc. Now with steadily increasing experience, opinions are becoming more standard and uniform.

## TIME AND POSITION FOR EXAMINATION

The X-ray examination of the appendix is usually a part of a complete gastrointestinal examination and is done at the usual intervals of six and twenty-four hours, and later, if necessary. At six hours when the barium meal is scattered in the coils of the ileum it is not so easy to see the appendix. If an examination is made for the appendix alone, the ideal time is twelve hours and upward. The examination must be extended as long as twenty-four or forty-eight hours or more, not only because the appendix is often more easily seen, but also to

note delay in emptying due to mechanical interference. The best position for fluoroscopic examination is with the patient lying on the back, as for the usual abdominal palpation. Special positions are rarely needed. The use of the fluoroscope is very valuable, in order to get down into the right lower quadrant and palpate the appendix to see if it is tender, and to get a good view of it by pushing the cecum and ileum out of the way, and to detect and judge the extent of adhesions.

#### DIRECT SIGNS

*Tenderness.* Tenderness is not strictly a roentgen sign; the value of the X-ray is in sharply localizing it and attaching it to some organ because we see the organ when we palpate it. Constant tenderness over the appendix (and not elsewhere) or on trying to move it is the best single sign of pathology. Most normal appendices are not sensitive and can be pushed about quite freely, depending on the length of the mesentery. Tenderness over the appendix when seen with the fluoroscope is far better than tenderness over McBurney's point on physical examination, which may be far from the appendix, even 5 or 6 inches or more. In some other conditions, for example sacro-iliac strain, the tenderness may remain fixed, may be in the same spot in both erect and horizontal positions while the appendix moves about freely with change of position, and has no relation to the tender spot.

The full, heavy cecum in atony and ptosis is often tender and we must not stress this tenderness too much unless the appendix itself is seen. If we find a tender cecum in a ptosis case and no appendix visible, we must not hastily conclude that the tenderness is due to a retrocecal appendix. These ptosis cases cause most of the mistakes in diagnosis and are most often operated upon needlessly. Tenderness is a very important sign, one of the most important, but it is subjective, and it should not be the main support of the diagnosis, but should be taken, if possible, with other signs, such as changes of form, adhesions, etc.

*Filling.* The filling of the appendix is important, but expert opinion varies greatly from George (1) who says that an appendix which does not fill is pathological to Skinner (2) who says that an adult appendix which does fill is pathological. These two extremes mean that an appendix according to George which does not fill has an obliterated lumen, and is therefore wrong; or according to Skinner that the appendix is the "abdominal tonsil," a lymphoid structure, that it

should normally and properly obliterate and keep out barium, and if it does not obliterate it is wrong. We must not be misled here, nearly all pathologists agree that obliteration is a sign of disease (fig. 8), that it occurs after repeated inflammations and deposits of scar tissue, and is not simply the disappearance of the lymphoid tissue of the growing animal.

I do not care to make the diagnosis of an obliterated or retrocecal appendix simply because it cannot be seen, for I believe many other appendices may not happen to be filled at the time of examination. Cohen (3) says the appendix may fill and empty several times during the passage of the same opaque meal, which is one reason why it is not always seen. We have found the appendix filled in more than one-half our cases. When seen, it can be studied and diagnosed, when not seen, little can be said about it. When an appendix is faintly outlined it may be due to partial filling with barium, not to obliteration of the appendix. The appendix is like the rest of the colon, and partial filling of one portion after a barium meal may be only accidental and temporary; not due to narrowing.

The kind of barium meal is worth mention. Some say that the buttermilk meal fills the appendix more frequently than the starch meal. I have used a starch gruel routine, and comparison in occasional cases with buttermilk, has given the same results. George (1) with a buttermilk meal gets 70 per cent filling, and Strom (4) in a recent paper with potato gruel reports over 70 per cent filling. The variation in figures given by different men probably represent more the degree of intensive study and care in examination, than the different quality of the meals.

The filling of the appendix may be irregular, interrupted, beaded, or segmental, or show fecal masses.

Simple segmentation or beading of the contents of the appendix deserves a word (figs. 4 and 5). It has been commonly mentioned in the past as a sign of disease in almost every paper read. What is it due to? We can only tell by physiological comparisons, by repeated examinations of the same case to show its constancy, and by comparisons with appendices at operation or autopsy. Peristaltic waves go into and come out of the appendix in the lower animals, and in man to some extent. This segmentation or separation of contents may be due to tonic contraction rings, such as are found in the colon of the rabbit and dog. Haustration is the rule in the colon in man, why may it not extend to the appendix? On the other hand the beading

may be due simply to the absorption of water and drying out of the contents of the appendix and separation of the barium meal into segments. No such anatomical change in the appendix is ever found at operation or autopsy; the process is probably purely physiological.

Filling around fecal masses in the appendix which show as oval light spots like peas in a pod, is often suggestive of disease (fig. 6). They can be found in almost anyone, but they are found most often and most markedly in appendices which do not empty well, and where the feces have dried up into little scybala. Such badly draining appendices often cause trouble.

*Emptying.* The normal appendix empties in twenty-four to thirty-six hours or more. There is probably a large normal variation up to a day or two, such as occurs in the rest of the colon. We are suspicious of poor drainage if the appendix remains filled much over thirty-six hours or after the cecum has emptied (figs. 1 and 2). Slow emptying is not an important sign unless accompanied by others, such as tenderness or changes in shape; or unless marked, two days or more. Adhesions may cause delay in emptying the cecum, but usually do not.

*Fixation* is important, especially if it involves one part of the appendix, the tip or median part, and causes kinking and deformity (fig. 2); this shows adhesions from previous inflammation. Mere bending of the appendix has no diagnostic value, for the appendix like other parts of the digestive tract varies greatly in contour and position within twenty-four hours. Fixation or kinking must be permanent, and not merely apparent or accidental to have any value in diagnosis. We must remember that there is considerable variety in the length of the mesentery of the appendix, and like the cecum some normal ones are freely mobile, while some are rather fixed. Adhesions naturally may involve also the cecum, ascending colon, transverse colon, ileum and pelvic organs, and cause fixation and deformity.

*Changes in shape.* Kinking and angulation are usually due to adhesions, narrowing, scar tissue and obliterative changes (figs. 4 and 7); irregular dilatation may be due to obstruction with delay in emptying and fermentation of contents (fig. 1). Simple large size is not dilatation, the appendix varies in diameter like the cecum and colon, as a result of muscular tone and personal peculiarity. There is so much change of form due to different positions and conditions of filling of the appendix that any change of form must be constant to have any diagnostic value as to the existence of stenosis, kinks, etc.

*Position.* This depends on the position of the cecum, which may be high above crest of ileum, or deep in pelvis, or in the median line, or even far to the left; little was known about this before the days of the X-ray. The position of the appendix is partly accidental, it is easily pushed about by palpation, or by the pressure of other organs, and is quite variable in the same case at different times if the appendix is free. The appendix is often near McBurney's point, but is often far from it, 5 or 6 inches or more. The clinician should remember this, the radiologist knows it well. The appendix may be retrocecal, and not seen till the cecum is empty, or it may be very obviously out of place, and fixed, bending upward toward the liver (fig. 6) or behind or outside the cecum (fig. 3); appendices so placed are usually diseased.

#### INDIRECT SIGNS

*Ileal stasis.* Slow emptying of the ileum with residues of the barium-meal, twelve to twenty-four hours or more, is the result of obstructive delay from adhesions. There are other reasons, however, for such delay; let me illustrate. In the X-ray report of a given case I find "considerable delay in emptying the ileum, no barium has reached the colon in six hours, considerable residue in the ileum at the end of twelve hours; diagnosis, probable adhesions in the ileocecal region, question of chronic appendicitis." I find that the patient is an old lady of seventy, who does not empty the ileum very well, neither would she run a mile very well. Emptying the ileum is a muscular effort. It depends on the muscular power and tone of the intestine. Delay here is due to ptosis and atony of the bowel, it is not obstructive, there is no question of appendicitis. In weak, sick, or old people there is often delay all along the line, the stomach, ileum and colon. Do not stress ileal stasis in such cases as these; in the atonic, ptotic cases are made most of the mistaken diagnoses, and most appendices taken out needlessly. The picture is iliac stasis, low, full, tender, slowly emptying cecum, sensitive nerves, pain or distress in the right iliac region; the diagnosis of chronic appendicitis is made; out comes the appendix, and the patient is no better. Sometimes there is a low grade inflammatory process in the appendix which also involves the terminal ileum, cecum and ascending colon, and there is little or no use in cutting off the appendix; in fact the adhesions after operation, the slower emptying of the right half of the colon may make the patient even worse than before.

*Indirect stomach signs* are spasm of the pylorus and duodenum with six-hour stomach residues after a barium meal. Spasm of the pyloric region occasionally occurs in "chronic appendicitis," but the spasm is variable and uncertain, and has little constant effect on function. In 100 cases published by the author (5) five years ago with chronic changes about appendix, one-half with adhesions in the ileocecal region and almost one-half (42 per cent) with ileal stasis of twelve hours or more, only 7 showed delay in emptying the stomach. Alvarez compares the intestine to a railroad under the block system where delay low down the line regularly holds up food for several blocks above. This is not always true; an irritating lesion in the lower bowel may or may not slow the progress of food coming toward it from above; depending on the character and degree of the irritation. Our clinical observation and experimental work in cats and men just referred to, all pointed definitely one way, showing, first, that delay in emptying the stomach is the exception not the rule in lesions of the lower bowel, and second, that a strong stimulus is needed from the lower bowel to slow the stomach. In short, pyloric and duodenal spasm and gastric residues are not good diagnostic signs of chronic changes in the appendix. They are only occasionally met with, say once in 12 to 15 cases.

*Incompetent ileocecal sphincter* has little relation to the appendix, and is too common in the absence of chronic appendicitis to have any diagnostic value.

*Adhesions* involving the ileum, colon and pelvic organs indicate congenital veils or local inflammation of any kind; previous appendicitis is a common cause.

(Lantern slides were shown illustrating X-ray signs, such as fixation, kinking, beading, changes in shape, abnormal position, lack of filling, slow emptying, and how often the appendix was far from McBurney's point. Some of the cases with these signs had been operated upon. In another series showing definite chronic changes, the appendix had been left in the abdomen for periods of one to four years after the X-ray examination, and the patients have remained well.

#### CLINICAL IMPORTANCE OF THE SIGNS

Are there any normal appendices in adults? I have read more than once that "pathologists teach us there are no normal appendices." Dr. F. B. Mallory, the pathologist at the Boston City Hospital, tells me that practically all the appendices taken out at operation for acute or chronic appendicitis at the hospital are pathological. Is this

simply the kind hearted pathologist backing up the surgeon, or does it mean that appendicitis is so common that all adult appendices are more or less pathological; no matter what you take out you are sure to find a bad one? How many normal appendices are found at autopsy? Dr. Mallory tells me that in routine autopsies in 4000 cases at the Boston City Hospital 95 per cent of the appendices were normal *in gross*, and showed no adhesions or kinking, no scar tissue or ulceration, no obliterative changes, no constriction, no deformity of shape, none of the signs which are easily discoverable by the X-ray. Three per cent showed adhesions, scar tissue or obliterative changes. Two per cent showed acute inflammation, gangrene or perforation. There were only 3 concretions in the 4000 cases. In short, when we get X-ray evidence of chronic changes in the appendix, they are not universal. They have importance as they are found in only about 5 per cent of adults.

The X-ray examination of the appendix is a very delicate method and discovers many things about the appendix, which were unsuspected and incidentally many things which may not trouble the patient much, such as poor mobility, small fecal masses, peculiar shapes, chronic obliteration of lumen, "beading," moderate delay in emptying. These were common signs in our group of cases, which were not operated. We have seen these patients going about for one to four years after the X-ray examination with practically no digestive symptoms whatever. The moral is that when these signs are discovered by the X-ray we must not make too much of them, consciously or unconsciously. Many persons will be helped by having a "chronic appendix" removed, many another will get no help at all.

"Chronic appendicitis" is not an entity. The changes vary greatly in degree and kind and in what should be done about them, all the way from the mild, harmless chronic obliterative type to the kinked, bulbous, tender badly draining type. The atrophic obliterated organ of middle life and beyond usually gives no symptoms and makes no important trouble.

Graphic X-ray evidence is always striking, and makes a great impression, and when the radiologist gives this graphic evidence to the family doctor, it would do no harm to remind him of these things. There is no organ so often removed without cause merely because of some anatomical change. Lahey (6) has recently called attention to the same thing from the surgical end. How often in taking out the appendix in a laparotomy done for other reasons the appendix is found

firmly bound by adhesions, so that the X-ray pictures would surely show fixation or poor filling, and yet there is no definite history of an attack, and no present illness. The wise surgeon will surely hesitate about removing an appendix on X-ray evidence alone without symptoms.

In our operative group we have always found a different group of signs, namely, constant tenderness of the appendix itself, sharp kinks with fixation, marked delay in emptying with tenderness, or a retro-cecal position with tenderness, and in addition the patients have given a history of local pain or tenderness. The diagnosis has been made chiefly by direct signs in the appendix itself, or by adhesions. The most important direct signs have been tenderness of the appendix, constant changes in shape, fixation, and abnormal position, and the less important have been the filling and emptying of the appendix, and signs of fecal residues. "Beading" is often normal.

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FIG. 1. BEADED, TENDER APPENDIX; REMAINS FILLED AFTER CECUM HAS LARGELY EMPTIED. APPENDECTOMY



FIG. 2. FIXED, IRREGULAR, TENDER APPENDIX; LUMEN NARROWED AT BASE; REMAINS FILLED AFTER CECUM IS EMPTIED. APPENDECTOMY



FIG. 3. APPENDIX POINTS UPWARD BEHIND CECUM WITH  
TIP ABOVE CREST OF ILEUM, TENDER.  
APPENDICETOMY



FIG. 4. APPENDIX SHOWS SOME FIXATION, ANGULATION  
AND BEADING, NOT TENDER. NO OPERATION; NO  
SYMPTOMS FOR FIVE YEARS AFTER X-RAY  
EXAMINATION



FIG. 5. APPENDIX SHOWS TYPICAL "BEADING," SOME FIX-  
ATION, NOT TENDER. NO OPERATION; NO SYMPTOMS  
TWO AND ONE-HALF YEARS AFTER X-RAY  
EXAMINATION



FIG. 6. APPENDIX SHOWS BARIUM FILLING AROUND FECAL  
MASSES "LIKE PEAS IN A POD;" TIP FIXED, NARROWING  
OF BASE, NOT TENDER. NO OPERATION; NO SYMPTOMS  
THREE YEARS AFTER X-RAY EXAMINATION



FIG. 7. APPENDIX CURLED UP, BEADED. MODERATELY TENDER. NO OPERATION; NO SYMPTOMS ONE AND ONE-HALF YEARS AFTER X-RAY EXAMINATION (OPERATION WAS EXPECTED IN THIS CASE)



FIG. 8. TYPICAL OBLITERATIVE CHANGES. APPENDIX VERY NARROW, MOBILE, NOT TENDER. NO OPERATION; NO SYMPTOMS TWO YEARS AFTER X-RAY EXAMINATION

## DISCUSSION

DR. ARMISTEAD C. CRUMP, New York City: There is one point that I should like to bring out, and that is that most of the cases have an atonic dilated colon. You will notice that the blood vessels are extremely dilated also with naturally a congestion of that part of the gut. Now the appendix is a part of the cecum and, in itself, is rather a poor piece of mechanics; and if congestion occurs in the colon, the congestion in the appendix is much worse. This congestion results in pain or tenderness centered in the iliac plexus. Taking out the appendix does not relieve the tenderness as the congestion remains. We have a wonderful follow-up service in the Presbyterian Hospital and quite a percentage of the patients have returned at variable times with the same old symptoms. My opinion is that the cecum is primarily the seat of the trouble. There is a dilation, atony and stasis of that organ. Exercise, diet, with plenty of water will do a lot in getting these patients well.

Regarding the patient that Dr. Lynch spoke of, there was no abdominal rigidity and only slight epigastric pain. It was hard to elicit any tenderness in the lower right quadrant. Her leucocytes were only about 12000, polynuclear cells being 80 per cent, but we decided that as she looked so septic the appendix had better come out. Just before the operation there was developed a slight relative rigidity in the lower right quadrant. The appendix was removed and found to be gangrenous.

DR. LEWIS G. COLE, New York City: This question that Dr. White has presented, it seems to me, is one of the most difficult that the roentgenologist has to meet. I do not believe that the diagnosis of chronic appendicitis ought to be made solely on X-ray findings. This opinion is at variance with statements that I have made concerning gastric ulcers and cancers and post pyloric ulcers; but we are now talking about the appendix. In all the plates that Dr. White showed, I could see no evidence one way or the other for leaving the appendices in or taking them out. George says that he can fill the appendix of 9 persons out of 10; and I believe that he is right. He did not say that he could keep it filled, but that he could fill it. Skinner's report would seem to be much at variance with that. He maintained that the appendix that was filled was pathological. He referred to one that remained filled, and probably retained that material for a considerable amount of time. I think that the retention of barium is an important pathological finding, when used in conjunction with the clinical findings. In the cases that Dr. White showed, all were probably twenty-four to forty-eight hour plates. How is the appendix going to empty until the cecum gets emptied? If the barium remains in there a longer period after the cecum is emptied, I believe that it begins to be of clinical significance. In my opinion, definitely deforming adhesions involving the left side of the cecum, and causing it to be constantly deformed are of more importance than any plate that Dr. White has shown. This roentgenological finding can only be obtained by an examination made with the cecum properly filled with an enema. Then it is an indication that there has been an inflammatory process in close proximity to the appendix, and this can only be used as confirmatory evidence in a case giving a clinical history of appendicitis.

DR. JOHN BRYANT, Boston : Just a word concerning one developmental aspect of this subject. Ten or twelve years ago, I heard Westenhofer state that from the point of view of the postmortem room, there were two types of appendices; those with conical bases and those with constricted bases. Westenhofer added, as is common knowledge, that the appendix with the conical tip represents an earlier and less differentiated stage of development than the appendix with the narrowed base. He made the unqualified statement that he had never seen a real acute appendicitis in a case in which the appendix was of the conical type, the obvious reason being that as the lumen was bigger at the base than at the tip, there was little chance of this lumen being closed by any inflammatory process. On the other hand, in a very large experience, the fulminating acute appendices which he remembered having seen were without exception of the narrow base type, the point being that but a small amount of additional constriction is necessary to convert such an appendix into a closed test-tube, within which bacteria find ideal conditions for multiplication and increased activity.

Westenhofer's observation has a certain amount of significance for clinical medicine in view of the fact that as others beside myself have shown, there is very frequent relationship between external body form and the character of the viscera. Thus, in regard to the shape of the appendix it may be stated that one may expect to find the long conical type of appendix associated with the long thin body form, whereas the constricted test-tube type of appendix is much more frequent in patients with the short thick-set type of body form. In a word, familial chronic non-fatal appendicitis occurs predominantly in families of asthenic or carnivorous habitus, whereas the serious and frequently fatal cases of fulminating appendicitis occur chiefly in families of hypersthenic or herbivorous habitus. Consequently when one is in reasonable doubt as to whether an asthenic has a beginning acute appendicitis or not, it is reasonably safe to postpone operation. On the other hand, should a hypersthenic present signs or symptoms which suggest even a reasonable probability of his having acute appendicitis, the appendix of this individual should be removed with the least avoidable delay.

I have personally seen many examples of the prevailing correctness of this proposition, and frequently make use of it from a diagnostic or prognostic point of view. This question of the relationship of external body type to visceral anatomy is therefore presented, not in the class of positive data such as temperature or blood counts, but as one additional factor in helping one to decide for or against the operation of appendectomy, should there remain, as often, a reasonable degree of doubt as to the existence of appendicitis after due consideration of all the usual scientific data.

DR. BURRILL B. CROHN, New York City: I should like to mention a paper, spoken of earlier this morning—a paper from German literature, on the examination of a large series of chronic appendicitis in which the appendix was removed and a clinical study made afterwards. The author comes to the conclusion that what we call chronic appendicitis should be called pseudo-appendicitis, on the ground that in many cases the appendix was removed and histological or gross study showed that certain changes had been taking place. By the histological or pathological study, the surgeon felt warranted in removing the appendix; and yet 70 to 80 per cent of the cases showed the same symptoms after operation as before, in spite of the fact

that sufficient pathological change was found to justify the operation. Therefore, he substitutes the name "pseudo-appendicitis" in the cases of this kind. In the cases where there is some real change, he suggests the name of chronic recurrent appendicitis. He also maintains that there is no such thing as a primary chronic inflammatory disease, but that all are cases of recurrent acute attacks of appendicitis.

DR. MILTON M. PORTIS, Chicago, Ill.: I was much surprised by Dr. White's quotation of Dr. Malloiy's statement that of 4000 cases examined, 95 per cent showed normal appendices. I should like to ask Dr. White whether those cases were examined carefully with the microscope—whether many sections were made, serial sections. It is astonishing, aside from that, that the gross examination showed that 95 per cent were normal. That upsets my previous conception.

DR. LOUIS M. GOMPERTZ, New Haven, Conn.: In the state of Dakota there is a law which inflicts a penalty of \$100 for every normal appendix removed. If that law existed in Massachusetts the State would soon become wealthy.

If operation was advised for the removal of the appendix on X-ray evidence alone, many unnecessary operations would be performed.

I very often have difficulty in keeping patients from operation against the advice of some X-ray man. I cannot agree with Dr. Lynch, however, that the X-ray has no value, but it must be taken in conjunction with the history and clinical evidence.

DR. D. HARLOW BROOKS, New York City: I should like very much to ask what Dr. White considers as a normal emptying time for the appendix. Various radiologists seem to differ very much on this point and it seems to me to be a very important point to determine if we are to value this factor in our diagnosis of chronic appendicitis.

Does Dr. White feel that the cecum should under normal conditions entirely empty itself of the barium before the appendix is clear? Does the evacuation of appendix and cecum occur synchronously under normal conditions?

As a clinician, I feel that too much importance should not be given to X-ray findings as regards the diagnosis of chronic appendicitis unless such findings seem to confirm those indicated by the clinical picture.

DR. JOHN A. LIGHTY, Pittsburgh, Pa.: A very important point is the aid which X-ray can give us in making the diagnosis of chronic appendicitis. I consider this diagnosis one of the most difficult in the abdominal cavity. It depends largely upon the clinical history, and where a satisfactory history of a previous attack cannot be obtained the diagnosis must always be doubtful. A great deal of our differences in opinion on the appendix depend on the very point which Dr. White has brought out; that is, our conceptions of the pathology of the appendix. The question is, Is there ever a chronic appendicitis without previous acute attacks? I believe that, as clinicians, we should hold to the idea that there is no chronic appendicitis without previous acute attacks. This may be a rather broad statement, but I think we frequently miss, in the history, the fact that previous attacks have occurred. In my own experience, I have questioned patients before the operation numbers of times and have been unable to get a satisfactory history.

After the operation when the patient was told that definite pathology was found, somehow or other he seemed to be able to recall a rather definite history of previous attacks. The acute attack may be apparently very mild but there may be considerable pathology following, so that the clinical history and the pathological findings will not always appear to agree.

In the X-ray findings I have been impressed particularly with the retention of bismuth in the appendix, say, after forty-eight or seventy-two hours. Fecal matter can no doubt remain in the appendix in the same way and can do considerable harm. One can imagine, therefore, that in a rather insidious way inflammation of the appendix may come on, but if there is fecal stasis there is also likely to be an acute exacerbation which we should attempt to interpret but which I fear we frequently overlook as being of little consequence.

DR. JEROME M. LYNCH, New York City: As far as my own personal experience goes, I do not think that the X-ray is at all helpful in the diagnosis. In most of the cases in which the diagnosis of appendicitis is made by the X-ray, you commonly find the appendix normal, or an appendix that is kinked. I think that one should rely on his own judgment and the clinical history in determining whether the appendix should be removed or not. I think that anyone who has seen many cases will bear me out in this, and I believe that Dr. Crump will also bear me out in this view. In one case, we took the appendix out because the man was going South and was afraid, as he had had a previous attack, that he might get an attack while down South. We found the appendix nearly gangrenous. I do not believe that you can always tell from the blood examination whether the patient needs an operation or not. One must be guided by one's judgment. I do not think that from an examination of the appendix, one can determine whether it is normal or not.

DR. JACOB KAUFMANN, New York City: Dr. White's conservative discussion of the clinical importance of the signs of changes in the appendix elicited by X-ray examination is very timely. The appendix has been made the scapegoat for so many ills that we must thoroughly investigate the value of signs which are considered to denote pathology of the appendix. I should like to take up for discussion the one sign usually relied on most in the diagnosis of so-called chronic appendicitis and that is tenderness. I would suggest to the roentgenologist that he employ a method which I have been using in clinical examinations. For a long time I have made it a routine practice to examine every patient whose appendix was removed, and have found that in the vast majority of these cases one can still find the same tenderness on pressure in the appendicular region. That shows that tenderness in the appendicular region need not originate in the appendix; so it would be well on roentgen examination of patients to see whether the surrounding tissues do not still show tenderness after the appendix was removed. I think that the tenderness is connected with the nerve plexus around the right iliac artery. You get the same tenderness often by pressure over the iliac artery of the left side and also over the abdominal aorta. If roentgenologists would systematically take up this method, the investigation would aid in clearing up this point.

DR. FRANKLIN W. WHITE, Boston (closing): It is impossible to speak of all the points raised. In answer to the question of Dr. Brooks, "What is slow emptying

of the appendix?" I do not think that we can set any definite time. We feel that it should be emptied in two or three days; and we get more and more suspicious, the longer the delay. The relation to the emptying of the cecum is important. If the appendix stays full after the cecum is empty, it suggests stasis.

With regard to Dr. Kaufman's point about tenderness in the appendix region, I think it makes a difference whether we are speaking of it from a clinical or a roentgenological standpoint. Right lower quadrant tenderness may mean many things, but if the appendix is seen with the fluoroscope and directly palpated, and found constantly tender, and there is no tenderness anywhere else; and if the tenderness moves with the appendix when the patient changes his position this is a far more important sign than general tenderness in the right iliac fossa. Dr. Mallory's high percentage (95 per cent) of grossly normal appendices found at autopsy in adults is certainly striking. He told me that they had not been able to section all the cases as they would have done if it were a case that had been operated on for appendicitis. My point was that gross changes in the appendix such as narrowing, kinking, adhesions, concretions, etc., which are just the kind of things that we can find with the X-ray, were absent in 95 per cent of adults coming to autopsy in a general hospital.

I cannot agree with Dr. Lynch that the X-ray has no value in the diagnosis of chronic changes in the appendix. I think that it has a great deal of value. It is a delicate method and does show clear signs of pathology, but it does not tell us what to do about them. There is our opportunity for clinical judgment.

I agree with Dr. Gompertz that we should not take out the appendix on X-ray evidence alone. I showed a series of cases with pathological changes in the appendix, in which it was left in the abdomen.

I agree with Dr. Cole that the diagnosis of chronic changes in the appendix should not be made on the X-ray examination alone, and also that deformities of the cecum and colon may be an important sign of appendix adhesions. A single plate of the appendix may not be diagnostic, but I could not arrange to show a series of plates of each case; it would have taken too long.

I think that the term "chronic appendicitis" is unfortunate. We do not have a true chronic inflammation, except in the rare tubercular appendicitis. I think that we should speak of chronic changes in the appendix.

# A PRELIMINARY REPORT ON THE TREATMENT OF CARCINOMA OF THE ESOPHAGUS WITH COLLOIDAL SELENIUM

ELMER B. FREEMAN

*From the Digestive Clinic of the Johns Hopkins Hospital*

The anatomical relations of the esophagus are such as to make the diagnosis of esophageal diseases very difficult and the treatment even more so. Also, when one considers the relations of the esophagus to the trachea and the thyroid in the neck, the left bronchus, the arch of the aorta and the heart in the chest, it becomes very apparent that the pathological conditions in these organs may, and frequently do, produce symptoms which are referred to the esophagus. It must also be born in mind that diseased conditions quite remote from the esophagus may reflexly produce esophageal symptoms. Therefore, before discussing the treatment of carcinoma of the esophagus with colloidal selenium, I desire to briefly mention some of the symptoms of esophageal diseases and the special methods of study employed in the differentiation of the same.

Usually the first manifestation of esophageal disease is dysphagia but frequently a lesion may have existed for some time before this symptom appears. This is true especially in carcinoma, while in spasmodic disease dysphagia is frequently an early symptom. When one considers that the esophagus is subject to marked mechanical irritations from coarse, irritating foods, to thermic irritation from hot and cold liquids and also to chemical irritations, it seems rather remarkable that pathological lesions do not occur more frequently; also, when it is remembered that the capacity of the normal esophagus is only 100 cc., that the lumen is quite small, that for comfort in the swallowing act we depend very much on the elasticity of the esophagus, that, if much narrowing occurs at any point, obstructive symptoms occur promptly.

While dysphagia is the first symptom to appear in the majority of cases, it may sometimes be preceded by a sensation of fullness or discomfort or even by pain, that is dull and aching or sometimes acute and sticking in character, either in the sternal or epigastric regions.

However, the slightest dysphagia or discomfort referred to the region of the esophagus should call for a very careful study, always bearing in mind that the symptoms referred to may be caused by pathological lesions quite remote from the esophagus. The dysphagia which is due to carcinoma is characterized in the beginning of the obstruction by difficulty in the swallowing of solid foods only but after the disease progresses and the obstruction increases, even liquids are swallowed with great difficulty. Finally, complete obstruction occurs when nothing will pass through the esophagus.

On the contrary, spasmodic disease is characterized by a sudden onset and symptoms of almost complete obstruction which in a short time give way to normal swallowing, to be followed again by a return of obstructive symptoms. In these cases the obstructive symptoms are quite as pronounced with liquids as with solid foods. Associated symptoms depend mostly on the degree of dysphagia present. As long as the patient is able to take food in sufficient quantity, the condition remains quite satisfactory, but when they are no longer able to obtain sufficient nourishment, they begin to show definite signs of malnutrition and anemia and in the carcinomatous cases, cachexia as well.

A routine physical examination gives us practically no information in regard to the esophagus, for one can palpate only a small portion of the esophagus above the thorax. This portion is so deeply located that even palpation is unsatisfactory. There is nothing to be made out by percussion and the information gained by auscultation is unessential, but by this method we do sometimes hear a sound during the act of swallowing. The sound is best heard between the ensiform cartilage and the left costal margin. It is delayed or absent in obstructive lesions of the esophagus.

While the routine physical examination of the esophagus gives us no positive findings, the routine physical examination of the chest as a whole is very valuable in helping to rule out other pathological conditions which frequently produce symptoms, by pressure and otherwise, which are referable to the esophagus. The most important of these are thoracic, aneurism, cardiac hypertrophy, enlarged bronchial glands, mediastinal growths and enlargement of the thyroid.

Due to the lack of information gained from routine examinations, it becomes necessary to resort to special methods of study. These are: sounding of the esophagus with a soft stomach tube or bougie, X-ray studies and esophagoscopic examination. All of these should be

resorted to when a patient complains of dysphagia, however mild, or any abnormal sensations referred to the region of the esophagus.

The stomach tube is a valuable means of locating an obstructive lesion in the esophagus as it is soft and there is no danger of injuring the esophagus when the tube is manipulated carefully. No effort should be made to pass the tube if obstruction is met. By this method obstruction can be definitely located and some idea of the degree of obstruction may be gained.

The most valuable means of sounding the esophagus is with an olive bougie, passed over a silk thread as a guide. With a silk thread properly anchored in the small intestine, one may pass different sized bougies and in a measure work out the amount of obstruction present. One should never attempt to pass bougies unguided for there is a great danger of making a false passage and puncturing the esophagus. This is especially true in spasmodic stenosis of the lower end of the esophagus in which there is pouching of the esophagus to the right, above the diaphragm. In these cases the unguided bougie does not enter the cardia but passes into the pouch which is frequently lower than the cardiac orifice and the depth to which the tube passes gives one the impression that it has passed into the stomach. Any force used in the manipulation of the sound is likely to traumatize the esophagus and some cases of puncture have been reported. When the stomach tube fails to enter the stomach and a large bougie (passed over a silk thread guide) enters without difficulty, you may be sure that the obstruction is due to spasm and not to organic disease. Some difficulty in passing a silk thread into the stomach is experienced by most patients but if the patient is instructed to swallow the first few feet very slowly, this will be overcome. In a few cases belladonna is helpful by overcoming the spasm. Not only can the degree of obstruction be measured but some information as to its character can be noted as well. In malignancy, one notes the marked resistance to the bougie and frequently the irritability and tortuosity of the canal, while in spasmodic stenosis there is a sort of elastic resistance, which, with gentle pressure, gives way and permits free passage of the instrument. This difference in resistance is of the utmost importance in differentiating spasmodic from organic disease.

A careful X-ray study offers more in the way of diagnosis than any other method of examination. In most cases it locates the pathological lesion and gives us valuable information as to its extent and character. Before an X-ray examination is made, it is very neces-

sary to empty the esophagus of its contents—otherwise, irregularities in outline and contour may be due to food and secretion retained in the esophagus. This is especially true in cicatricial stenosis and spasmodic disease. In cicatricial stenosis what is from an X-ray standpoint apparently a complete obstruction, may be found to be patulous after all of the food remnants have been removed. While evidence of obstruction with irregularity in contour is evidence of malignancy, it must also be born in mind that malignancy, especially of the lower end of the esophagus, may occur without irregularity.

It is also perfectly well understood that chronic spasm in the lower end of the esophagus, while usually having a smooth outline, may show irregularity. Further, it must be born in mind that spasmodic stenosis and carcinoma of the esophagus may occur together, and from an X-ray standpoint are very difficult to differentiate. While the evidence this method supplies is very valuable, it is sometimes misleading. Therefore, the X-ray interpretation must be made in the light of the clinical history, physical examination and the other special methods of study. Thus, I believe that to arrive at an accurate diagnosis, it is necessary in many cases to use all of the available methods of examination.

Most internists and many surgeons look upon esophagoscopy as a dangerous and difficult procedure and oftentimes unnecessary. We believe that there is very little danger and no difficulty in making an examination, if one has a thoroughly trained assistant and we are convinced that this method of study is very helpful in differentiating esophageal diseases.

We have adopted a very simple procedure which has been quite successful. It is as follows: For two days previous to the examination the patient is given belladonna in dosage sufficient to cause some dryness of the throat. This, of course, lessens very much the secretion in the throat and esophagus and at the same time helps to overcome any spasm that may be associated with the pathological condition. In most cases, no further preparation is necessary but in a few it is necessary to give  $\frac{1}{4}$  grain morphine and  $\frac{1}{160}$  grain atropine, hypodermically, half an hour before the examination, also to make two or three local applications of 10 per cent solution of cocaine to the pharynx. After the patient is thus prepared, we proceed to empty the esophagus by using one of the newer suction apparatuses, to which is attached a small sized stomach tube. This tube is passed down to the obstruction and then the contents aspirated. By this method

we are reasonably sure that all contents have been removed. The patient is now seated on a low stool, the head held in the proper position and the esophagoscope is passed through the right pyriform sinus.

To make a careful esophagosopic examination quickly one must have an assistant who is thoroughly familiar with the routine, as a faulty position of the head makes the introduction of the instrument impossible and any attempt to pass the same is fraught with great danger to the patient. It is also very important to have the esophagus free from food and secretion—otherwise, secretion obstructs the view by coming into the lower end of the tube, making further examination futile. But if the patient is properly prepared and you have a thoroughly trained assistant, you will find that esophagoscopy is not difficult and will be a valuable aid to diagnosis in a group of cases that are sometimes very difficult.

In the clinic we have followed this simple routine as outlined and have found it, as far as our work is concerned, most satisfactory. We never make an esophagosopic examination until the patient has had a routine physical and X-ray study. This procedure is followed so as to rule out, as far as possible, all pathological lesions that might be producing esophageal symptoms by pressure which, of course, contraindicates any mechanical manipulation of the esophagus. With these conditions ruled out one may proceed with safety.

While the X-ray is our most valuable means of diagnosis, still this method of study cannot replace endoscopy, by means of which one may obtain a direct view of the pathological lesion. Surely, if all patients with symptoms referred to the esophagus were seen early by the endoscopist, some pathological conditions could be diagnosed at a time when satisfactory treatment could be given.

We have seen small ulcers in the mucosa that were very difficult to diagnosis endoscopically. However, the endoscopic appearance of most lesions, when once definitely established, is very characteristic. These small ulcers when situated near the cardia, I am convinced, are responsible for some cases of spasm of the lower end of the esophagus and if these cases were subjected to an early routine endoscopic study, those that were due to ulceration could be diagnosed and treated before atony and dilatation occur. When one finds ulceration going on in the esophagus, then it must be decided whether or not the lesion is a simple ulcer or one due to tuberculosis, lues or malignant disease. Cases of peptic ulcer have been reported, also those due to actinomycosis.

In simple ulcer one usually obtains a history of some mechanical, thermal or chemical irritation but in a few cases there will be nothing in the history to indicate an injury to the mucosa. The simple or benign ulcer occurs most frequently at the anatomical narrowings of the esophagus which are also the most frequent location of malignant disease. Practically all of those due to traumatic injury from foreign bodies occur in the upper third of the esophagus, as 90 per cent of the foreign bodies lodge in the region of the cricoid cartilage which is the narrowest part of the esophagus. The healing of a simple ulcer (unless very superficial) causes stenosis of the esophagus in varying degrees and the endoscopic appearance of the same depends on the degree of stenosis, the duration of the disease and the character of the irritant that has caused the injury. The most extensive injuries are caused by caustic alkalies.

If the stenosis is slight, the mucosa appears normal and there is no dilatation above the stricture. If the stenosis is of high degree, chronic inflammatory changes with or without superficial ulceration or abrasions will be noted in the wall of the esophagus above the stricture. Marked dilatation will also be noted. Cicatricial tissue in the esophagus is always paler than the normal mucosa. It may appear almost white. The appearance of the stricture depends upon the location of the scar. In some cases, a linear scar will be seen with a drawing-in of the wall of the esophagus. In other instances, the scar is seen to cross one side and cause a flattening of the lumen. In others, the scar causes a polypoid protrusion into the lumen. In many cases, the scar tissue almost encircles the esophagus, causing an annular stricture.

Esophagoscopy adds much to our knowledge in spasmodic disease of the esophagus. I believe that, in spasm of the upper end and early in spasm of the lower end, it offers more than any other method of study. By other methods it is difficult to make a differential diagnosis between spasmodic and organic stenosis of the upper end of the esophagus. An endoscopic study permits direct inspection of the diseased area which usually establishes the diagnosis.

In spasmodic stenosis occurring at the lower end of the esophagus, early in the disease, the endoscopic appearance may not differ from the normal. Late in the disease, the esophagus is very much dilated, the walls are atonic, the mucosal folds obliterated, the respiratory movements absent, the mucous membrane grayish in color, showing evidence of chronic inflammation with, in many cases, superficial ulceration.

The esophagoscopic appearance of cancer varies greatly according to the stage in which the examination is made and whether the growth is primarily in the esophagus or secondary to malignant disease in the adjacent viscera.

Jackson states that he has every reason to believe that the very early stage of esophageal cancer occurs as a leukoplakia in at least a few instances but that the opportunities for early esophagoscopy in cancer are so rare that there is no means of determining the frequency of such an onset. He states that, in these cases, the mucosa appears as though it had been burned with silver nitrate. In other instances, an early manifestation of malignancy is sub-mucosal infiltration which gives a sensation of increased resistance in the esophageal wall. This is always the early finding when the carcinoma involves the gullet from without; in these cases the mucous membrane remains apparently normal for a long time.

As the disease progresses, other local changes are noted, as irregularity in the lumen, obliteration of the normal mucosal creases and folds and the absence of inspiratory and expiratory movements.

Later on, definite polypoid protrusions into the lumen, with consequent marked irregularity in the outline of the growth are noted. At this stage, there is definite congestion of the mucous membrane and, in many cases, a beginning ulcerative degeneration of the growth. After ulceration has occurred, the appearance of the ulcer is not entirely dependent upon the carcinoma, but is partly caused by secondary infection with pyogenic organisms. A malignant ulcer is usually irregular in outline, the surrounding mucosa much inflamed, the surface granular and slightly elevated. It bleeds easily when sponged. Definite areas of necrosis may be found in the more fungoid and cauliflower types of neoplasms. Late in the disease, one is impressed by the marked rigidity of the diseased area, the tortuosity of the lumen, the ease with which bleeding occurs, the absence of scar tissue and the small amount of dilatation above the growth.

Endoscopically, a malignant ulcer may be confused with one due to lues or tuberculosis. In luetic ulceration, the following characteristics usually are seen: the edge of the ulcer is elevated and surrounded by markedly inflamed mucosa; the surface is frequently depressed; granulation tissue is not excessive and there is very little bleeding when the ulcer is sponged, although the surrounding mucous membrane may seem very much inflamed. The tuberculous ulcer is superficial, rather anemic in appearance; the granulation tissue is not

excessive; the edges are not elevated; the surrounding mucous membrane is not congested and the surface does not bleed easily when sponged.

As yet thoracic surgery has done but little for cancer of the esophagus. Gastrostomy as a palliative measure prevents starvation but a radical operation for cancer of the esophagus has been attempted only by a comparatively few surgeons and even in their hands it has not been very successful. However, with the recent advances being made in this branch of surgery, we believe that in the near future surgery will offer much to these patients.

Radium has offered more than any other method of treatment and, in the recent literature, some reported cures are to be found. Also much good has been done in a palliative way. However, with radium the problem of giving a dose large enough to penetrate the tumor mass sufficiently to destroy the cancerous cells in the outer zones without injuring normal tissue cells is still unsettled and if the cells in the outer zones do not receive a lethal dose, they may be stimulated to greater activity, with the result that a slowly growing malignant tumor may be made to grow very rapidly.

If the attack against malignancy could be made through the blood stream, then it would be possible to bring the therapeutic agent into contact with all of the malignant cells.

Wassermann, working with mouse cancer, in 1911 and 1912, set out to solve the following problem: Is it possible to influence therapeutically by intravenous injection without injury to other cells of the organism, a rapidly growing tumor? Can one find a chemical compound which, after intravenous injection, will automatically leave the blood stream, attach itself to the tumor cells and destroy them?

The first experiments here to be reported were based upon the work of Gosio (*Zeitschrift fur Hygiene*, Band 51, 1905) with natrium telluricum and natrium selenicum, which give a red or black precipitate with living cells (due to reduction of salt to metallic state), but no precipitate with dead cells.

A. V. Wassermann readily proved that local injections (most easily with telluricum) would destroy carcinoma. However, this treatment is useless for tumor of inaccessible or unknown location, and intravenous injections are needed.

At first intravenous injections (tail veins of mice) were without effect and it was thought advisable to provide for increased diffusibility by the addition of other chemical groups. For this purpose

were tried several hundred compounds with the fluorescein dyes (eosin, erythrosin, cyanosin) combined with selenium and telluricum. These compounds were most unstable and most difficult to prepare. Finally, a combination of selenium and eosin was found to be very active. This substance is soluble in water and can be injected without fatal results in amounts up to 2.5 mgm. for a normal mouse of the average size (15 grams). Following the injections the whole mouse becomes red.

Using this dose, in mice with malignant neoplasm, these results were obtained: doses I and II, no visible changes; doses III and IV, the tumor softened to a fluctuating cyst and began to be resorbed; after dose V and VI the tumor gave the sensation of an empty sac and rapidly disappeared.

With too rapid resorption the animals died from toxemia. After complete cure, no recurrences developed, but with incomplete treatment, early recurrences were the rule.

This treatment was uniformly efficacious with several strains of carcinoma (3 from Ehrlich, 1 from Schilling, and 2 spontaneous strains) and was even more effective with a strain of sarcoma from Ehrlich's laboratory. Of these tumors, none disappeared without treatment. Autopsies showed a red stained mass, or detritus, or fluid with pale or colorless tissue about it.

One can easily prove by putting small pieces of fresh tissue in the thermostat in selenium solution that the selenium is reduced by the living tissue and precipitated in the metallic state (fine black granules) particularly near the nucleus where the need for oxygen is greatest, but not reduced by the dead tissue. These results can be readily repeated with local injections into carcinoma in living animals. With intravenous injections of eosin-selenium, the action is mainly upon the cell-nuclei and there is very little leucocytic infiltration. The spleen of these animals frequently shows a deposit of this pigment, or even small masses of tumor detritus which have been carried there by the blood stream. This detritus is sometimes found also in the liver and lungs of animals which are responding to repeated injections. In a few cases nephritis occurred with necrosis of the epithelium.

The authors give warning that human tumors may not be susceptible to this drug, but state that mouse carcinoma can be cured by eosin-selenium in intravenous injection.

The poisonous nature of the preparation of eosin-selenium used rendered its direct application to human beings too hazardous. This

difficulty was surmounted by the production of a colloidal suspension of selenium, the toxicity of which has been shown by animal experimentation to be negligible. Thus the way was opened to the therapeutic application of the remedy.

E. Watson Williams, in the *British Journal of Surgery*, 1920-1921, makes the following statement:

In many inoperable cases, the results obtained with selenium compare favorably with any known method of attacking a malignant growth by the way of the blood stream and are apparently equal to those of radiotherapy. All malignant tissue is effected, however inaccessible. As purely palliative treatment, the drying of ulcers and abolition of pain is a marked advantage.

He reported 18 cases treated, with apparent arrest of the malignant process in 6. Diminution in the size of the tumor could be verified in 5 more. The condition of the remaining ones was not conclusive. That is, apparent arrest and relief of symptoms followed treatment in 1 out of 3 cases and in the whole series, the majority experienced considerable benefit.

From the possibilities of Wassermann's experimental work and the recent clinical report of good results obtained by Williams, we decided to carry out some observations with selenium in carcinoma of the esophagus. We selected this group of cases because we believe them to be the most hopeless of all.

Colloidal selenium may be administered subcutaneously, intramuscularly or intravenously. When given subcutaneously, there is quite a little local reaction and the results have not been satisfactory. The intramuscular method gives results similar to the intravenous route, though less promptly.

In the cases under observation, we are using the intravenous method. The general reaction is not alarming if one observes carefully the effect of the initial dose. The injection may be given in the office or the outpatient department of a hospital and the patient allowed to go home half an hour later. Repeated injections may be given into the same vein. If, for any reason, some of the solution gets into the subcutaneous tissue while giving the injection, the local reaction is negligible. The initial dose we have been giving is 3 cc. This is followed on the second day by 5 cc. If no reaction follows, this dose is given three times a week throughout the treatment. In this sized dosage there has been no cause for the least anxiety. So far, we have not given more than twenty treatments.

Following the intravenous injections in most cases there is, in a short time, a slight anaphylactoid shock as evidenced by frequent pulse, weakness, nausea and occasionally a chill. There may be also increased difficulty in swallowing for a short time. This is thought by Williams to be due to an increased blood supply to the growth and this to be secondary to a local deposit of selenium following the initial reaction which usually occurs within the first half hour. There is in six to eight hours a general reaction which continues for ten to twelve hours. During this time there is some rise in temperature, increased pulse rate, headache and general depression. Williams thinks this is due to absorption into the circulation of material destroyed by the drug. The initial shock or the general reaction which occurs during the first twenty-four hours usually only occurs after the first few injections or when the drug is given in very large and frequent doses.

In the cases under observation, there seems to be some improvement in symptoms. There has been relief from pain, dysphagia has become less marked, loss of weight has at least been temporarily controlled and, in one case, a very marked gain in weight has been noted. There has been marked psychic improvement which may be due entirely to the fact that some definite line of treatment is being carried out. However, I believe the improvement is partly due to the relief from pain which usually occurs after the first few injections. Also, the dysphagia is lessened and the patient is able to take more nourishment.

While the treatments are being carried out, careful esophagoscopic observations are made from time to time. The appearance of the presenting part of the growth is noted; first, to determine whether or not there is evidence of increased blood supply to the part; second, if any sloughing or necrosis has occurred; third, if the esophagus has become more flexible or more patulous. We try to make these observations twenty-four hours after the selenium has been given. These cases are also studied by X-ray methods to determine, if possible, any change in the size and contour of the growth and also to note whether or not obstructive signs are becoming less marked.

As an adjunct to the treatment, dilatation is carried on by bougies being passed over a silk thread as a guide. This method of passing bougies prevents injury to the esophagus which is quite likely to occur when they are passed blindly. The passing of bougies blindly is mentioned only to be condemned.

Our group of cases is too small to draw definite conclusions from, but we do feel justified in making the statement that in a certain group of cases this method of treatment may be useful.

It would seem from the literature that so far very little has been done with selenium in the treatment of carcinoma and especially in a large number of cases, conclusions might be worked out which would serve as a working basis in the future. We hope to follow out the work in a sufficient number of cases to try to formulate logical conclusions, not claiming at the present time anything more than a palliative treatment to be used in a certain group of cases which, up to the present time, have been, if not entirely hopeless, almost so.

We do not know whether or not the results obtained from this method of treatment will compare favorably with those where radium is used but it must be borne in mind that for certain reasons, radium is not available to all patients suffering with carcinoma. If further observations on the use of selenium will establish its therapeutic value, then we have a method of treatment which is available for all cases.

The surgical treatment is to be given preference over all other methods when the technical difficulties of the same have been overcome, but up to the present time these difficulties have been so great that only a few surgeons have perfected a technique which justifies them in attempting radical operations for carcinoma of the esophagus. I do believe, however, with an earlier diagnosis, which will be made possible when the profession as a whole recognizes the importance of an early, careful study of all patients with symptoms referable to the esophagus, that some of these patients will be referred to the surgeon in time for operative treatment. This is especially true, I believe, of carcinoma of the lower end of the esophagus. I have seen one such case in the past year. In this patient the tumor was small, situated just above the diaphragm with apparently very little infiltration of the wall of the esophagus.

I am well aware of the fact that in many cases symptoms are overlooked by the patient or do not occur until the disease is well advanced and little can be hoped for in this group. But, on the other hand, many times the presenting symptom of dysphagia or pain is overlooked by the physician or attributed to hysteria or some other nervous manifestation until the disease is well established and the most important time for study and treatment is passed.

## DISCUSSION

DR. THOMAS R. BROWN, Baltimore: I was very much interested in this work, as I happened to have 2 cases in which there was a specific action of the colloid material on the growth. Of course, we are dealing with a discouraging group of cases, which should be diagnosed relatively early, because progressive dysphagia is present in the majority of cases of carcinoma of the esophagus. These cases show the symptoms early, and do not metastasize early. For that reason, except in the practice of a very few surgeons, the method has not been attempted. I feel that it is rather a blot on surgery that in this group of cases, no more attempt has been made at radical removal. Up to now, the difficulty has been great, because of the prevalence of secondary infections which are preventable. We have had one of our old surgical residents, a Pittsburgh man, at work during an entire year on the subject in the Johns Hopkins Laboratory; and I believe, from what he told me, that there is a basis of hope. It does not prove anything at all yet; but it is merely an honest attempt, on the part of an extremely able man, Wassermann, to make an attack on the malignant growths. Anyone who saw him while he was living in Berlin, as I did, will realize that as far as the neoplasms on mice are concerned, there is a definite action on the growths in mice. The (colloid?) material will be abstracted almost entirely from the blood stream and found in the tumor itself. It is an attempt at direct treatment by means of chemotherapy on the cancer cell. I attempted to employ the method when I came back from Germany; and in 2 cases, striking phenomena were noted. Shortly after the administration, there was a great increase in tenderness in the neoplasm itself, which suggested that the colloid material went directly from the blood stream into the growth. In the second place, there was a marked shrinking in the growth in a certain number of cases. There was, however, no case in which the method did not finally prove unsuccessful. There was a lessening of the symptoms and a shrinking of the growth, but in no case did it do more than prolong life and palliate the symptoms. The significant thing, however, is that it showed that there is a possibility, shown experimentally, and with much less foundation clinically, for certain materials, colloid materials, to have an enzymic effect on certain tissues—that chemotherapy may attack cancer cells. We hope that the surgeons will preclude the possibility of utilizing this method, however, by their success.

DR. A. MCGLANNAN, Baltimore: We surgeons hope that the internists will so prove themselves expert with this method of treating cancer. We tried it in the case of one patient with recurrent cancer of the glands of the neck after carcinoma of the mouth. The patient was a physician, and anxious to try it. We gave him two doses; but the general reaction was so violent after the second dose that he would not allow us to try any more. He preferred to go on with a knowledge of his hopeless condition from cancer, to going through another such reaction.

One point in the surgery of carcinoma of the esophagus has been, in my experience, overlooked many times: the expedient of gastrostomy has been postponed until late in the course of the disease. In the last two years, I had 5 successive patients come to me for gastrostomy when they were really hopeless risks. We felt, however, that we should do what we could to give relief. In all, death occurred within a month to six-weeks after the operation. They were debilitated and starved out people. Direct surgical attack on the tumor is a matter for surgeons,

yet gastrostomy is a simple operation that can be done by anyone who is competent to open the abdomen and it is a good thing to do in order to relieve the patient while he is being treated by some other means.

DR. ARMISTED C. CRUMP, New York City: There is one thing that I should like to bring up at this time, the symptomatology of the condition itself. Unfortunately, the class of patients that have that symptomatology and go to a doctor are, in my experience, very poor people. Most of them are clinic cases. The first symptom that generally occurs in these cases is dysphagia. When it gets to the point of pain, as a rule, it is pretty far progressed. Only once have I had a case that came with the symptom, not of trouble in deglutition, but that of a feeling of distress immediately on swallowing. Unfortunately such cases linger in the general medical part of the dispensary, and are treated by giving them a mixture for dyspepsia. The wise thing, if they get into the hands of the general practitioner, is to keep to the idea of being suspicious. In middle life, there is a condition of the esophagus in which the symptom comes on during swallowing. When the patients have had the condition a month or two and there is the onset of pain, they are more difficult to treat.

One patient that I had in the ward was seventy-eight years old. It would have been foolish to attempt an operation. I dilated the esophagus. I do not know how long he lived. We could not get a follow up.

DR. ALLEN A. JONES, Buffalo, N. Y.: May I ask, Dr. Freeman, what you said about the frequency and the dosage? I think you said that you introduced 3 cc. of the remedy. How often?

DR. ELMER B. FREEMAN, Baltimore (closing): In the beginning of the treatment, 3 cc. are given every third day for four doses. If no reaction occurs, the dose is then increased to 5 cc. three times a week. We have never had a severe reaction. Some patients, however, have had a chill followed by a moderate rise in temperature. We are not claiming anything for the treatment; we are only trying to work out some form of treatment for a disease, which up to the present time, has been considered almost hopeless. If further observations seem to indicate that the method of treatment is worth while, we will make a clinical report of the cases so treated. While under treatment, most of the patients apparently had less difficulty in swallowing, and were more comfortable in every way. How much the improvement in symptoms was due to the improved psychic condition of the patient, I am unable to say.

# THE RÔLE OF SPASTICITY IN DISEASES OF THE DIGESTIVE TRACT (CASE OF VISCERAL TETANY, CAUSING ACUTE CHOLANGITIS AND PANCREATITIS)

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It is the object of this paper to contribute to a better understanding of the rôle which spasticity may play in diseases of the digestive tract.

The knowledge of abdominal spasticity is not new: The writings of older clinicians show that they were familiar with the phenomenon. Roentgenology, however, demonstrated its great frequency and taught us to differentiate more sharply its various forms and types and their relation to tone, peristalsis and segmentation (hypermotility, hyperperistalsis, tonic and tetanic contractions). Granted that the appearance of spasticity has generally been recognized, its interpretation and valuation is often far from satisfactory.

Its frequent occurrence with local conditions, such as traumatic lesions, stones, acute inflammations, ulcerations and cancer, led to the more common explanation of spasticity as a manifestation of localized character, a plausible explanation on account of the automatic activity of the ganglia implanted in the digestive tube. And yet operative, as well as postmortem inspection, found the lesion often at a different spot than the location of the spasm would indicate: ulcer or cancer at the cardia with spastic obstruction in the upper part of the oesophagus; ulcer or cancer at the smaller curvature with pronounced spasm of the uninvolved cardia; gall-stones and cholecystitis associated with true gastrospasm or with tonic contractions in the colon and sigmoid. The spasm in parts distant from the lesion is produced reflexly over the paths of the vegetative nerves which regulate the coöperation of the different sections of the digestive tube. Reflex spasm may also originate from organic lesions outside the alimentary canal, from diseases in the central nervous system, the pelvic and other organs. These two types, local and reflex spasm, shall not be further discussed here since they predominate in the interpretation of spasticity at present. I rather intend to emphasize a fact

too often overlooked, that spasticity, eventually of a very high degree, may, as the result of systemic derangements occur without the presence of a local lesion.

#### AUTONOMOUS SPASTICITY

The conception has been entertained long ago, that spasticity may present itself as part manifestation of conditions which we are still accustomed to class as general neuroses.

In his classic essay on "peristaltic unrest" Kussmaul (1) attributed the painful and visible hyperperistalsis of the stomach and the bowels, to a highly increased irritability of the peristaltic nerves in neurotic individuals, at the same time stating with regret that the term motor neurosis did not convey much meaning. Under his influence Fleiner (2) defined spastic constipation.

The modern study of the working of the vegetative nerves, greatly stimulated since the description of vagotomy by Eppinger and Hess (3), has considerably advanced the understanding of spasticity of systemic origin.

We have learned that normal activity of the digestive apparatus requires a harmonious interplay of the vagus and the sympathetic and that it depends on a maintenance of steady tonus in the two systems. However, the habitual tone of the vegetative nerves varies greatly as to degree in different individuals, and even in the same person it varies at different times according to his constitutional make-up. Constitution has been aptly defined as a person's characteristic way of reacting to internal and external stimuli. Coined on this basis, the term "spastic constitution" is suggestive. Such definitions make it easier to comprehend that the same type of local lesion may provoke frequent and severe spasm in one person, and rarer and milder spasm in another; and further, that a local lesion may exist without causing spasm, that for instance, stones in a gall-bladder may remain dormant, often for a long period of time, until something occurs in the system which provokes spasm in the biliary ducts and with it "an attack." Even the structural narrowing caused by cancer often becomes evident only at a time when spasm is superimposed and produces the symptom-complex of obstruction. Persons with habitually high irritability of the vegetative nerves seem to be disposed to a further increase of tone by anything affecting their physical and mental balance, foremost of all by fatigue after physical, mental, or emotional taxation.

Once we have reached this viewpoint, the association of local lesions with spasticity may appear in a different light. While in certain conditions the local lesion must be held chiefly responsible for bringing on the spasm, for other conditions the question may well be asked whether the spasm is not primarily the result of systemic influences, and instead of being an effect, is not, in fact, a causative element the development of the lesion.

#### SPASTICITY AS A CAUSATIVE FACTOR

The valuation of spasticity as a causative factor has been extensively dwelt upon in the discussion on the pathogenesis of peptic ulcer. Experimental investigators, pathologists and clinicians join in the contention that the primary lesion, that is, the miliary ecchymoses, are caused by spasm of the muscularis mucosae which constricts the small end-arteries and leads to ischemia and extravasation. When a strong peptic secretion is present, which like the muscular spasm is the result of excessive vagus tone, the poorly nourished part of the mucosa in the area of ecchymoses is digested, and we have an erosion. The development of an erosion into an ulcer and its further growth are possible, however, only when there is more than temporary over-irritation of the vagus nerve. It requires a distinct disposition, which manifests itself by a chronic state of increased vagus tone, bringing on prolonged periods of hypersecretion and spasm not only of the muscularis mucosae, but also of the larger muscular coat and eventually of the pyloric sphincter. The regular recurrence of spasm with each meal prevents the healing of the ulcer and makes for its chronicity. Once developed, the ulcer as a source of irritation may further increase the vagus tone thus creating a vicious circle. From the very start and through the whole course of the ulcerative process we see spasticity as an important causative element.

If we accept these views which offer a plausible theory of the mechanism in the development of (certain types of) peptic ulcer, we still have to answer the question: What causes the spasticity or rather the excessive vagus tone, which is at the bottom of the ulcerative process? At present the opinion prevails that it is provoked reflexly by a local lesion somewhere else in the system, especially by a diseased appendix, in short that peptic ulcer is a "secondary disease" (Rössle, 4). I am not ready to subscribe to this opinion, having seen too many cases where the ulcerative process recurred or even started only after the removal of the appendix, and also hesitate on account of similar ex-

periences after other operations. On the contrary, I think clinical observations justify the contention, that increased irritability of the vagus with consequent spasm may be produced directly by the action of systemic influences, (diets, etc.) metabolic disorders, auto-intoxication, lead- or tobacco-poisoning and other factors: As I pointed out to you four years ago (5) mental and emotional overtaxation, probably by way of disordered endocrine influences may be the principal causative element in the development of excessive vagus tone, spasticity and active periods of ulceration. Only when we realize the importance of systemic factors are we able to explain the well known periodicity of peptic ulcer. This periodicity is but a confirmation of the axiom, that disease is not static, but a functional disorder with stages of development. The local reaction is merely a manifestation in a certain region of predisposing systemic agencies.

It is this point which I wish to stress today, that spasticity resulting from systemic conditions may be a primary causative factor in peptic ulcer, and in a similar manner in other local lesions in the digestive tract. There is at present too much inclination to look for a direct causal relation between the different diseases in the stomach, intestines and gall-bladder, making one the provocative agent of the other. It is certainly true that, for instance, a diseased appendix may reflexly cause disorders of the gastric function. Such possibilities, however, are far too often generalized. It occurs to me that we may arrive at another view by investigating, whether each one of simultaneous but different local lesions met with in the same person, was not originally the outcome of a systemic disorder. This view would rather tend to coördinate them, and would minimize the influence, which after their initial development they may have had upon each other.

Without entering now upon a discussion of the nature of the underlying general condition, I repeat that spasticity caused by systemic upsets, may prove an important factor here. By approaching these problems in a changed mental attitude, clinical syndromes may assume to the observer a different significance.

Since abdominal surgery demonstrated the frequency of acute infections especially in the appendix and gall-bladder, conceptions of abdominal pathology have been strongly influenced by the picture of inflammation. The result was that not only in acute, but likewise in chronic conditions, objective as well as subjective symptoms, such as tenderness and pain, were too readily accepted as signs of inflam-

mation. This, for instance, is clearly seen in the poorly defined syndrome of "chronic appendicitis," the diagnosis of which is principally based on the evidence of spontaneous and pressure pain in the appendicular region. The fact that in a high percentage of cases thus diagnosed as chronic appendicitis, the same spontaneous and pressure pains recur after appendectomy, proves that the pain did not originate in the removed appendix. When not caused by another local lesion or not of a purely neuralgic character, the pain in many of these cases is the result of uncomplicated enterospasm. We do well to keep in mind, that in the digestive tube, as elsewhere, spasm by itself may produce pain, eventually of severe character and of long duration.

For a while the idea of the existence of true nervous gastrospasm and of enterospasm was actually ridiculed; it has come into its own again since the roentgen-ray visualized such spasms. It is significant that a surgeon, Liek (6) in Danzig, after a thorough study of the literature and of his own vast material, rejects the popular syndrome called chronic appendicitis. He adopts instead the term pseudo-appendicitis, attributing the pain in most of these cases to nervous enterospasm.

When the spasm is localized at the appendix, it may cause stasis, bacterial growth and inflammation. In such an event inflammation follows the spasm. Acute appendicitis is frequently preceded by attacks of "appendicular colic" without fever and without changes in the blood picture.

A similar situation may arise in the biliary tract. In a paper (7) read before this association nearly twenty years ago, I stated that spasm at the opening of the common duct, by causing biliary stasis, may lead to the formation of gall stones, to calculous attacks and to infections and inflammations of the bile ducts and of the gall-bladder. Dr. Arpad Gerster (8) independently arrived at similar conclusions in a paper in which he dealt with the causes of unsuccessful surgery in disorders of the gall ducts. And Dr. Samuel Meltzer (9) discussed this topic thoroughly with you at a symposium on diseases of the gall-bladder in 1916. That the same underlying condition of autonomous spasticity should create such entirely different products as, for instance, a clean-cut peptic ulcer or a purulent inflammation is not so strange if we consider the peculiar qualities of the gastric secretion, which not only digests poorly nourished parts of the gastro-duodenal wall but also destroys bacteria.

Clinical experiences over a course of years have convinced me, that spasm alone, due to systemic upsets, more frequently than is generally conceded may account for severe abdominal pain; furthermore that it may become the primary causative factor in the development of abdominal diseases including local inflammations. As an illustration I present the history of a patient whom I had occasion to observe together with his physician (Dr. A. L. Garbat) and with other consultants.

Patient, L. M., male, fifty-five years old, grew up under trying circumstances: from childhood on had to do very hard work, while for many years he was provided with poor food. At an early age dental decay set in and was neglected, so that eight years ago, all teeth had to be extracted. From early days whenever his stomach was upset frequent and long attacks of hiccups appeared. He always had a tendency to "indigestion" with heartburn and sour eructations. Appetite was usually good, bowels were constipated.

No acute attacks of pain ever occurred until the present trouble.

In 1906 at the age of thirty-four, pleurisy with effusion was contracted. Was tapped twice with removal of 92 ounces of fluid. Tubercle bacilli were present in the sputum. Involvement of left upper lobe and right apex were found. After prolonged stay and treatment at Saranac, Adirondacks, was discharged with general improvement and gain of weight. No signs of an active process showed themselves until 1913, when an attack of fever and cough required his return to Saranac. After this the picture remained that of an inactive, stationary fibrosis of the left upper lobe and of the right apex. Patient returned regularly to Saranac during the summer months, and several times during the year for a few weeks whenever he felt run down. On these occasions more cough and more sputum were noted with tubercle bacilli. During the rest of the year, the high strung, energetic, short and moderately nourished man worked steadily and over long hours as boss of a barber-shop in a big hotel, where he had "to handle from 600 to 700 customers a day."

In the summer of 1920, while at Saranac, the first attack of severe epigastric pain set in. It penetrated to the back, was taken for an acute indigestion, although the possibility of gall stones was also considered. The second attack came on June 5 1921: severe pain on right side, the entire right side was rigid and very tender, so that it was difficult to differentiate between gall-bladder and appendix. Temperature up to 103; while blood count was 12,000, 84 per cent polynuclear, 16 per cent lymphocytes. Light jaundice. Fever and tenderness gradually disappearing. While in bed for about three weeks undergoing a modified "Carlsbad cure" with application of heat to the right upper side, a sudden attack of very severe pain on the left side set in, radiating into the scrotum and left thigh, bearing all the ear-marks of a renal colic. No red blood cells or concretions were found in the urine. Was greatly benefited by the Carlsbad treatment. He went to the mountains for the whole summer and remained free of pain until September, 1921, when renewed attacks of very severe pain in the upper abdomen set in, radiating to the middle of the back and to the right shoulder. They became more frequent,

finally occurring every day. The pain was usually of such a severe character that no medication except morphine hypodermatically would give relief. At no time were fever or jaundice present. The X-ray showed stones in the gall-bladder.

October 5, 1921, Dr. John Erdman removed the gall-bladder with 77 stones and 5 stones from the common duct. The gall-bladder was thickened, firmly adherent to the liver and to the surrounding tissues and was separated with great difficulty. No signs of acute inflammation were found. Cultures from the gall-bladder were sterile. Free flow of bile was secured by drainage. Uneventful recovery until the twelfth day after the operation (one day after removal of the drainage tube from the bile duct) when the patient suffered again an attack of very severe abdominal pain of the same character as before. A week later he had a similar attack. From then on the attacks occurred more frequently, finally every day and even two or three times within twenty-four hours. The pain, excruciatingly severe, varied in its location. At times it was felt in the middle of the abdomen, radiating towards the back or the chest and in the latter event causing severe dyspnea; at other times it would start in the right upper side radiating towards the groin. Not infrequently the paroxysm was preceded by painful peristalsis and gaseous borborygmi. On several occasions particularly with the onset of phosphaturia (see below) very severe pain occurred on the left side, shooting into the bladder. It was associated either with great difficulty to pass urine, or with very frequent voiding of small quantities of urine. This dysuria was observed at other times with clear urine.

The paroxysms came on at irregular intervals, at any time during the day or the night. They were not in any way related to meals, were occasionally alleviated by an enema and alvine evacuation, and were, as a rule of such a severity, that none of the many drugs tried would give relief except morphine hypodermatically administered.

With numerous attacks occurring over a period of about four months no fever or any other signs of inflammation were ever observed.

*Blood Picture* was that of secondary anemia, for instance, January 12, 1922, hemoglobin 75 per cent, red blood cells 4,368,000, white blood cells 7200, polymorphonuclears 73 per cent, lymphocytes 27 per cent, Wassermann negative.

Blood pressure low, 90 to 100 over 40. No disorders of the heart.

Fluoroscopic combined with serial roentgenographic examination of the gall-bladder region, stomach and duodenum done on November 21, 1921, did not reveal any evidence of stones in the biliary ducts. The report of the roentgenologist continues as follows:

"We gave the patient the usual barium meal, and examined him in all different positions, both erect and prone. We were at once struck by the very vigorous hyperperistalsis, marked writhing contractions were seen almost immediately and continued throughout the entire observation. On manipulation under the screen, evidence of adhesions binding down the prepyloric region of the stomach and the first portion of the duodenum. On the upper border of the bulbous there is a persistent defect.

"In post-operative cases, one has great difficulty in deciding what is producing such a deformity to the outline of the first portion of the duodenum. It must be either one of two things, adhesions or true ulceration. Considering the character of the shadows, the persistency of the deformity, and the fact that we had such very marked hyperperistalsis, it would seem to be some ulceration."

After an examination of the entire urinary tract on December 13, the report reads:

"I fail to find any evidence of nephrolithiasis, nor do we find any shadows that are even suspicious of a stone in either ureter. In the bladder, well down toward the symphysis, we see some irregular shadows which suggest calcareous deposits in close relation to the prostate.

"The left kidney seems somewhat enlarged but is normal in form and position. The right kidney is not clearly outlined owing to the presence of gas and fecal matter in the proximal colon.

"If you will refer to my report of Mr. M's examination of November 21 you will note that I referred especially to the presence of a very vigorous hyperperistalsis. I also reported a distinct deformity to the first portion of the duodenum and in my conclusion stated that I believe the man has a duodenal ulcer."

Gastric analysis after test-breakfast showed nothing abnormal: Free acid, 36; total acidity, 58. No occult blood in feces.

Stool as a rule very scanty, dry hard scybala, often passed only with great difficulty, denoting spastic constipation. At other times the stool was copious, mushy, acid, reminding one of a pancreatic stool, without, however, showing impaired digestion of fat, starches or meat.

Duodenal drainage on January 27, 1922; tube inserted at night; next morning with fasting stomach after application of 50 cc. of 20 per cent magnesium sulphate solution, immediate free flow of bile. First portion dark and thick (absence of gall-bladder!); during the following six hours about 500 cc. of bile were gained, light yellow, viscid, without sediment, showing on microscopic examination neither leucocytes or epithelial cells, nor crystals. Culture sterile.

Whenever I had occasion to see the patient, I was impressed by a marked tendency to spastic conditions. At various examinations abdominal muscles between the attacks were found contracted and rigid; during attacks the whole abdominal wall appeared board-like in tetanic contraction. Having his attention aroused, patient reported, that at this period during meals, he frequently had to drop fork or knife on account of cramps in his hands. When walking he often had to stop for a while to let pass severe cramps in calves or feet. Even when in bed the abdominal paroxysm was frequently preceded or followed by cramps in calves or feet, often of very great severity. At one time (middle of January, 1922) he felt severe pains in the little fingers and in the big toes. I have already mentioned the spastic bladder condition.

This impression of spasmodophilia was strengthened by findings of disordered calcium metabolism. While the urine as a rule showed nothing abnormal, during the month of December, 1921, for several weeks regularly, and in January, 1922, for a while intermittently, the urine was of peculiar and unusual character; freshly passed, the urine was turbid, strongly alkaline, had a very pungent odor of ammonia and a very heavy whitish powdery sediment, which the microscope revealed as consisting partly of amorphous material, to a greater extent, however, of triple phosphate crystals, while there were no cellular elements pointing to an inflammation of the urinary tract, and no indication of bacterial fermentation.<sup>1</sup>

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<sup>1</sup> Regarding the relation of phosphaturia to disordered calcium metabolism I refer to f. Ueber, Ernährungs und Stoffwechsel Krankheiten, 2nd edition, 1914, Chapter on Phosphaturia, p. 469.

At a time when the urine had been clear for a while, a study of the calcium metabolism by Dr. A. Bernhard, pathological chemist to the Lenox Hill Hospital, showed the following:

*Metabolic study of calcium*

|                                       |                   | CALCIUM AS<br>CaO |
|---------------------------------------|-------------------|-------------------|
| January 20 to January 21, 1922        |                   |                   |
|                                       |                   | grams             |
| Excretion                             | Urine (1000 cc.)  | 0.335             |
|                                       | Stool (256 grams) | 3.910             |
| Total                                 |                   | 4.245             |
| Intake (in food; exclusive milk diet) |                   | 3.900             |
| Loss of calcium during first day      |                   | 0.345             |
| January 21 to January 22, 1922        |                   |                   |
| Excretion                             | Urine (1450 cc.)  | 0.529             |
|                                       | Stool (249 grams) | 8.510             |
| Total                                 |                   | 9.039             |
| Intake (in food)                      |                   | 2.500             |
| Loss of calcium during second day     |                   | 6.539             |

Average daily calcium excretion in urine X for two days..... 0.432

Average daily calcium excretion in feces XX for two days..... 6.210

X: Normal excretion of Ca in urine 0.1-0.4 grams.

XX: Calcium elimination by feces normally between 60 to 90 per cent of the amount ingested, while in this case, with only 6.40 grams intake, there was an excretion of 12.42 grams, instead of 5.7 grams at the utmost.

*Report on chemical examination of blood*

*Normal*

|   |             |       |
|---|-------------|-------|
| Urea N.....                                   | (12.0-15.0) | 22.0  |
| Creatine.....                                 | (0.5-2.0)   | 1.0   |
| Uric acid.....                                | (1.0-2.5)   | 5.0   |
| Sugar, per cent.....                          | (0.08-1.20) | 0.080 |
| Calcium 6.2 mgm. per 100 cc. (normal 5.3-6.8) |             |       |

From a report of the status of the nervous system taken on January 21, 1922, I quote the following:

"Pupils equal, both react to light, accommodation and consensually. There is no disorder in the territory of the facial nerve. The tongue is coated, but protrudes in the median line, and shows no atrophy or fibrillation. No motor disorder

manifest in the entire body; no sensory disturbance except as indicated below. Deep reflexes markedly increased; superficial reflexes are normal except for an absence of the upper and lower abdominal reflex on both sides. Some hypalgesia over the left lower abdominal region. Mechanical excitability of the muscles of both forearms, markedly increased with a pronounced idio-muscular ridge easily produced. Mechanical excitability of right lower abdominal muscles markedly increased. Electrical reactions normal except in the muscles of the right lower abdominal region (scar territory) in which there is a reduced faradic excitability and anodal closure contractions greater than cathodal closure contractions, in the abdominal muscles due to the incision made in the right lower abdominal region.

Although this examination showed no electrical hyperexcitability of the peripheral nerves at the time of the examination, the various manifestations of tetanoid contractions of peripheral muscles before and after this examination suggested the probability that we were dealing here with a similar hyperexcitability of the abdominal vegetative nerves with consequent spasm and pain, as were indicated by the finding of intense gastric hyperperistalsis during the roentgen examinations. Attempts to correct this condition by dietetic and various medical treatments, including the administration of parathyroid and calcium chloride by mouth, proved futile, so that, when at the end of January the paroxysms of pain became more frequent and for several days required two to three morphine injections within twenty-four hours, we decided to open the abdominal cavity once more, considering the possibility that a remaining gall stone, cancer, or another undiagnosed local lesion might be the provocative agent. At the wish of the patient the operation was set for a certain date, when a complete change of the picture necessitated an earlier operative interference. On February 2 with an atrocious attack of pain particularly severe in the back, fever up to 103 and pronounced jaundice were observed; the urine was full of bile, the stool clay colored (careful searching of the stool for a number of days failing to find stones).

*Operation February 3, 1922:* Dr. John Erdman found dense adhesions at the site of the removed gall-bladder impinging upon the common duct. The common duct was readily exposed and found fairly dilated. Upon opening the common duct, a considerable amount of purulent material was extruded, a scoop and catheter, the latter the size of no. 16 French readily passed through into the duodenum. No stones in common or hepatic ducts; no signs of peptic ulcer. Head of pancreas considerably swollen and indurated; choledochostomy was completed by sewing the catheter into the duct and drainage established. Culture of common duct content showed pure *Coli comm.*

For two days an uneventful course, except for frequent vomiting. From the third day on the spastic tendencies returned once more. For a number of days very frequent painful urination of clear urine was present and a much more annoying symptom, that is singultus, which, with short intervals, continued during eleven days, greatly sapping the patient's strength and unrelieved by any sedative, atropin, alkalies, acids and other drugs. Morphine injection gave relief for a few hours, likewise gastric lavage, which was for several days performed two and three times a day, always removing large quantities of fluid with turbid masses. An attempt to employ duodenal tubing miscarried, the tip remaining in the stomach.

Considering the evidence of disordered calcium metabolism I advised intravenous injections of calcium chlorid, 5 cc. or a 10 per cent solution, for three days given

twice a day, then one a day, then every other day and finally twice a week. Altogether 20 injections. After the first injection of calcium chlorid, the singultus stopped, appetite returned and food intake was tolerated. From now on rapid recovery followed, patient regaining strength, remaining free from all discomfort and adding up to the middle of April, 20 pounds in weight. It is worthy of note that with all the very severe disturbances the lung condition remained stationary and inactive.

*Commentary.* The inflammation of the dilated common duct found at operation was of recent date, with fever and jaundice of about twenty-four hours duration, while only a week before duodenal drainage had produced a free flow of bile carrying no signs of inflammation. Since no stones were discovered and Vater's papilla proved wide open, it is not stretching imagination too far, to consider the acute cholangitis a result of severe spasm involving the sphincter of the common duct, which also caused acute pancreatitis by a flow of infected bile into the pancreas.

Spasticity is a predominant feature in the history of this patient. From childhood on pronounced tendency to hiccups existed and during the period of observation frequent cramps were observed in various parts of the voluntary neuro-muscular apparatus.

Combined with spasmophilia we found evidence of disordered lime metabolism. At one time there was an unusual type of phosphaturia and at another period an increased discharge of lime from the intestines.

#### DISORDERED CALCIUM METABOLISM AND VISCERAL TETANY

The association of disturbed calcium metabolism and tetany after experimental and operative removal of the parathyroids led to the conception, that a change in calcium content causes the increased irritability of the neuro-muscular apparatus. There is still a great divergence of opinion regarding the actual relation of parathyroid deficiency and of disordered calcium metabolism—also regarding the character of the calcium disorder. Some authors assert that in this deficiency the calcium content of the blood is low, while others maintain that the total amount of calcium present in the blood is not as important, as is the quantity of calcium present as free calcium (10). However that may be, when the parathyroids are involved, we can well imagine, that functional parathyroid insufficiency (of whatever origin) may account for temporary states, both of disordered calcium metabolism and of tetany (analogous to paroxysmal tachycardia). Barker and Sprunt (11) claim that temporary tetany may occur without defective functioning of the parathyroid glands. But let chemical changes making for spasticity once develop in the system—be this an effect or not of parathyroid malfunction—these changes may not only increase the irritability of the voluntary neuro-muscular apparatus, but may in a similar way affect the vegetative nerves—a condi-

tion for which the term "visceral tetany" has been suggested. In a very interesting study Melchior (12) discusses the very great variability of the picture, which visceral tetany may present according to which part of the vegetative nervous system is particularly involved at a given time.

In our case the spasm occurring shortly before the onset of the acute cholangitis and pancreatitis must have involved Oddi's muscle. Whether during the preceding months the severe abdominal paroxysms were confined to the same locality is very difficult to determine. The varying location and irradiation of the pain point to a changing location of the spasm, for which the picture of renal colic with the onset of phosphaturia gives an illustration. It is possible that the roentgen examination during attacks might have visualized the seat of spasm at a given time. Where this was done it yielded interesting results. Melchior in the paper just quoted reproduces the roentgenogram of a patient suffering from gall stones (later removed by operation), which taken during an attack of pain shows a true gastrosplasm, the middle portion of the stomach being tightly contracted into a sausage shaped form. Three-quarters of an hour later the same picture was seen. A roentgenogram taken two days later during a pain free period revealed a normal non-ptotic hookformed stomach with good tone and with a completely filled out middle portion and antrum. This and similar experiences by Melchior and others should guard us against drawing from roentgenograms taken between attacks too close conclusions regarding the seat of pain and spasm.

#### VISCERAL TETANY AND PULMONARY TUBERCULOSIS

There is one more interesting feature in the history of this case, the combination of visceral tetany with pulmonary tuberculosis. It is a well known fact, that pulmonary tuberculosis is frequently associated with states of gastric irritation and with peptic ulcer. Singer (13) recently reported postmortem findings of peptic ulcer with pulmonary tuberculosis. They showed degeneration and neurotic atrophy of the vagus. Singer considers the anatomical changes of the vagus a product of the tuberculous process. In cases still devoid of manifestations of a lung involvement, he attributes the earlier state of functional vagus derangement to a tuberculous disposition. These cases present only symptoms of gastric irritation and Singer designates the gastric syndrome the sign of latent tuberculosis. One may gain a different aspect of the combination, by considering the possibility,

that both, the lung and the gastric disease, are the result of the same underlying general disturbance. The same constitutional make-up, the so-called asthenic habitus, predisposes equally to pulmonary tuberculosis and to derangements of the vegetative nerves. It is possible, that here also disordered calcium metabolism plays a part. We have just discussed its relation to visceral tetany and it may be added that French clinicians lay great stress on demineralization, especially upon the increased discharge of calcium and magnesia as one of the basal factors in the development of pulmonary tuberculosis. One may perhaps find some support for this conception in the favorable reports of the treatment of tuberculous conditions by the systematic intravenous injection of calcium chlorid, solutions (14) the same as those which were employed in our case.

#### CALCIUM THERAPY

Finally a few remarks about calcium therapy. I frequently use calcium in the treatment of irritative disorders of the digestive organs, employing large doses of lime water, calcium lactate, the various calcium phosphate preparations, from among which, for a while I used to favor the tribasic calcium phosphate, specially prepared, but it is difficult to procure. During the last few years I have been giving, with preference calcium chlorid, a teaspoonful of a 20 per cent solution of crystallized calcium chlorid in distilled water, to be taken in half a tumbler of water in the course of the principal meals. I have seen some favorable results, for example, in periodical painful gastric hyperacidity, which in certain cases was promptly relieved by the calcium solution. I advise patients subject to periodical hyperacidity and to periods of active ulceration to continue the calcium medication over a course of months, expecting its prophylactic effect in the same way as it is supposed to act as a preventive of hay fever (15). The beneficial effect of a long continued milk diet in peptic ulcer and other irritative disorders of the digestive tract is probably in part due to the high calcium content of the milk.

In our case we employed before the second operation both milk diet and the calcium chlorid solution, although only for a short period. They failed to make any impression upon the tendency to these severe paroxysms. After the second operation, however, the intravenous calcium administration seemed to stop the very annoying hiccup and the associated retroperistalsis of the upper digestive tract. I have no intention of overstating the importance of a single observation,

but this experience should certainly encourage further trials of intravenous calcium therapy in cases of visceral tetany.

Lime therapy is empirical and will remain so until we know more about calcium metabolism and its disturbances. So far our knowledge is scanty. Here is a large field for investigation with the vista, that modern chemistry will establish a solid foundation for the study of calcium metabolism, disorders of which may prove to be the underlying constitutional derangement in the development of local diseases of the digestive tract, as gout is in acute inflammations of joints and diabetes in gangrene.

I hope that the report of this case may arouse further interest in the investigation of disordered calcium metabolism, visceral tetany and their relation to local diseases of the digestive organs.

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## DISCUSSION

DR. MAX EINHORN, New York City: I do not like to pass this paper unnoticed. I think that most of us will thank Dr. Kaufman for his remarks indicating that spasticity and vagotonia play a great part in producing the symptoms in these diseases. He expressed the opinion that ulcer of the stomach is produced more often in case of nervous phenomena, an opinion which has become quite prevalent. I think, too, that nervous phenomena in spastic conditions can favor the production of ulcer; but I do not think that we can ascribe all the ulcers to that cause alone. I think that there must be some other things that are also causative factors in the production of ulcer. I fully agree with Dr. Kaufman that functional disturbances that are protracted are apt to bring on, ultimately, organic troubles.

With regard to the case that he has so ably described, I may be pardoned if I say that I was present at a meeting during which the same case was brought up by an associate of Dr. Kaufman's, and it was found that the patient suffered with various tuberculous lesions in the lungs. I think that this has some influence on the condition being of a spastic nature. I wanted to add this, because Dr. Kaufman omitted that. I am very grateful to him for presenting this paper.

JOSEPH SAILER: I should like to cite 2 cases that appear to bear some relation to Dr. Kaufman's paper. One was an old gentleman who suffered from diarrhea. No treatment seemed to have any effect. An appendostomy was done and the colon washed without any beneficial relief whatever. At the autopsy complete destruction of both suprarenal glands was found and we concluded that the depression of the sympathetic system had resulted in hyperperistalsis which was accountable for his diarrhea. The usual signs of Addison's disease were absent excepting a very low blood pressure.

The second case was one of obstinate constipation. The patient was a young married woman of rather an emotional type. The X-ray showed no evidence whatever of peristaltic waves in the gastrointestinal tract. Dr. Pancoast who studied her on a number of successive days stated that it was the only case of this kind that he had ever observed. It seemed possible that there was some depression in the autonomic system or in the musculature of the intestinal tract. She very promptly recovered on small doses of eserine which would indicate that the muscles were involved and not the autonomic system.

DR. FENTON B. TURCK, New York City: Dr. Kaufman called our attention to one of the most important objective symptoms of the digestive tract, namely, spasticity. It takes its place beside that frequently associated phenomena known as atony. A fundamental principle of biology, physiology and pathology is involved in this subject of muscle contraction, and we must consider the causative factors that are involved. I know of no one of Dr. Kaufman's intellectual attainments who is more ready to consider ideas which differ from his own viewpoint. One criticism I would make, however, is that this condition of muscle spasticity is claimed to be neurogenic in origin.

This psycho-neurogenic idea impregnates the whole of medicine, especially in those conditions where causes are unknown. For years war has been waged between the supporters of the myogenic and neurogenic origin of muscle action.

When His (1893) and Hooker (1911) found rythmical contraction occurred in non-striated muscle in embryos before the muscle tissue was invaded by nerve cells, it gave impetus to further studies.

Burrows (1912) and Lewis (1915) found in tissue cultures that independent rythmical contraction occurred entirely separate from nerve supply. From my own studies in tissue cultures and in the animals, I have concluded that the normal activator of muscle contracture is the extract of the tissues itself, and that in excess these tissue extracts act as specific poisons and cause spastic contractures or atony. Both conditions may be found in the same animal.

There is involved here, not only causes of contraction but also fatigue of muscle in which tetany-like condition of fatigue is pronounced. The clinical application of the scientific facts found on experiments, I presented before this society in 1903, on "A study of fatigue of gastric muscle." Also a further discussion in the Journal of the American Medical Association, 1904 (March 26), and Medical Record, October 7, 1905. My own studies have led me to conclude that spastic conditions are brought about by tissue toxins acting directly on muscle cells. My studies in shock led me to consider that anaphylaxis and spastic contractures are not a nerve reaction, but a direct cellular response to the tissue toxin which is autochthonous. Allergy from food intoxication appears to be of similar nature when foreign material becomes sensitized by homologous tissue extracts of the individual. The clinical importance of this viewpoint has recently been made more clear since the basis of the neurogenic theories have failed to receive experimental support. The reliance placed on De Bois findings of muscle tone depending on sympathetic innervation, has given way since it was not substantiated by two workers and though contrary to Langelans theoretical reasoning, we must admit that both vagus and sympathetic control is absolutely unproved. This was more recently confirmed by Herman Myers' work on spastic conditions of the esophagus in *Grenzgebieten de Medicin und Chirurgie* (1922, B. 34, H. 4). All central control is denied. There may be some peripheral coördinating values to consider in the nerve supply, but the chief factors as a basis resides within muscle cells. The tissue extracts play the dominant rôle in the biology, physiology and pathology of the muscle tissue of the digestive tract.

DR. JOHN BRYANT, Boston, Mass.: I should like to express my appreciation of the truth of Dr. Kaufman's remarks concerning the influence of purely mental or nervous conditions upon the intestinal tract. I have many opportunities to study this reflex effect and there is no doubt that in consequence we may frequently get high degrees of spasticity. I have for years followed one patient who has had recurrent attacks of spasticity following upon three or four days of especially severe mental commotion. In this case the spasticity is evidenced by severe local pain and greatly increased peristalsis, the pain when at its worst being often sufficient to require serious contemplation by the surgeon of the necessity for exploratory laparotomy; to date, these attacks have always subsided and I presume they always will, but during their maximum intensity it is very unpleasant not only for the patient but for every one else concerned.

Dr. Kaufman mentioned pseudo-appendicitis. Doubtless he knows as others do that so-called chronic appendicitis is frequently due merely to an unusual degree of distension of the cecum by gas or ingested air, as indicated by the ex-

tremely tympanitic note upon percussion. The mere pointing out of this chain of events, with proof by percussion, is often sufficient to relieve the nervous anxiety of the patient to an extent adequate to relieve pain.

Another item which should be mentioned in this connection, is focal infection. One sees many patients who have had their tonsils "removed" by some of the older methods. These patients frequently have a good deal of tonsil tissue remaining, a condition which from the point of view of symptoms is often disastrous. This is because the scar tissue from the incomplete operation prevents the normal discharge from the tonsillar crypts into the throat; therefore the patient gets by absorption the entire benefit of whatever trouble there may be within the remaining tonsil tissue. One often finds marked degrees of intestinal spasticity present in these patients who have been tonsillectomized by the older incomplete methods, and in view of the fact that the intestinal spasticity is usually much improved after removal of the remaining tonsil tissue, it is fair to assume that there may be some connection between the removal of this tonsil tissue and the relief of the intestinal spasticity.

DR. JACOB KAUFMANN, New York City (closing): Of course, I did not mean to say that over-irritation of the vegetative nerves is the only factor which enters into the development of the condition; but it is one of many, and a very important one.

In regard to Dr. Einhorn's question of the lung condition of my patient, that question is thoroughly discussed in my paper. Dr. Lyon will also find the details of calcium administration in my paper.

Regarding Dr. Turck's remark about undue importance being given to the influence of the mind, I hope that in spite of the vagaries of the Viennese school he is not willing to eliminate the influence of the mind altogether. After all, the influence of mental and emotional activity is important in all that happens. It is just this point, which differentiates the observation of a human being from that of an animal. And I should like to point out very sharply that clinical observations on human beings may be as much true scientific work as those in the laboratory, provided the observation in either case is made with proper amount of critic. As far as the influence of the disturbance on the muscle itself is concerned, I stated in my paper that spasticity often is of a local character. I advisedly in my paper used the expression "neuro-muscular apparatus," thereby indicating that both the nerve and the muscle may become more irritable as the result of the chemical changes which make for spasticity. The main point, which I wanted to bring out in the paper is this: that we must study more thoroughly systemic disorders. I do not think that we shall make much progress unless we go into the investigation of the general disorders, which are the underlying cause of local lesion.

# PEPTIC ULCER WITH DEFORMITIES OF THE VISCUS, EVIDENCED BY X-RAYS, CHANGED FOR THE BETTER BY TREATMENT

MAX EINHORN

*The Lenox Hill Hospital, New York*

The treatment of peptic ulcer is still a mooted question. The majority of surgeons lay claim to the entire field for themselves, while most clinicians believe that gastric and duodenal ulcers in a general way should be treated medically, except where there are special indications for surgery. Penetrating or callous ulcers of the stomach, or duodenum, which nowadays can be frequently demonstrated by the X-rays, have been considered by both physicians and surgeons alike as requiring surgical aid. For it was not believed possible or even probable that such far advanced pathological changes which showed marked deformities, easily recognizable by the X-rays, could be restored to a normal state of health by medical means.

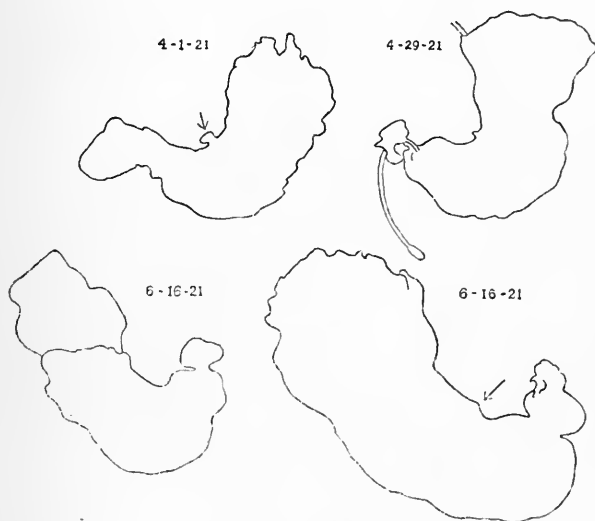
The doubt in the efficacy of medical treatment of peptic ulcers can be easily seen from the following statements made by Sir Berkeley Moynihan (1), who expresses himself as follows:

We ask for a series of cases in which the physician, the surgeon, and the radiographer are agreed that a gastric ulcer is present, in which medical treatment has been tried, in which all are confident that a healing of the ulcer has taken place, and that the healed ulcer has not again broken down. . . . Unless evidence can be produced to show that the real chronic gastric ulcer, not the phantom which wrongly bears its name, can be healed by medical treatment, the surgeon who in the diagnosis of this condition walks by sight and not by faith, is entitled to state that his methods alone can secure the end desired. At present I do not know of any such evidence I can accept.

The present paper deals with peptic ulcers in which constant deformities were visible by X-rays; in 5 there was a typical penetrating (callous) ulcer of the lesser curvature, in 1 a penetrating ulcer of the pylorus, and in 6 constant deformities of the cap. Surgeons, physicians, and radiographers would all unite in making a positive diagnosis of advanced peptic ulcer in these cases—they therefore fulfill the de-

mands of Sir Berkeley Moynihan—and they show the possibility of being treated successfully by medical means, with a restitution of almost normal conditions with regard to the configuration of the stomach and duodenum, as evidenced by the X-ray examinations.

Before discussing the cases I would like to mention that Hamburger (2), of Chicago, called attention to the utility of frequent X-ray examinations in cases of gastric ulcer recognizable by X-rays, in order to ascertain whether the treatment is of benefit or not. The same able clinician demonstrated a case of gastric ulcer with gradual improvement and subsequent favorable changes in the X-ray pic-



CASE L. K.

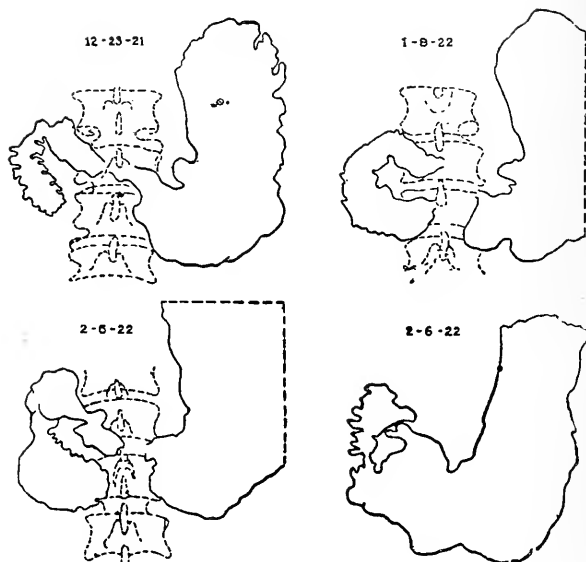
FIG. 1. L. K. April 1, 1921. Before treatment. Pouch of Haudek's niche distinctly visible.

FIG. 2. April 29, 1921. The pouch has diminished to about  $1/10$  its original size; the tube is still visible lying in the duodenum.

FIG. 3. June 16, 1921. Five weeks later. The niche has disappeared.

FIG. 4. June 16, 1921. Same. Prone.

tures. I have frequently shown that a positive string test has become negative in about 50 per cent of the cases after a two weeks' treatment of duodenal alimentation. Deformities visible by X-rays are not present in all cases of peptic ulcer. When they exist they certainly bear the best testimony to the presence of a far advanced ulcer, and if after treatment they are missing this circumstance appears to be the best evidence of a change for the better, if not of a cure.



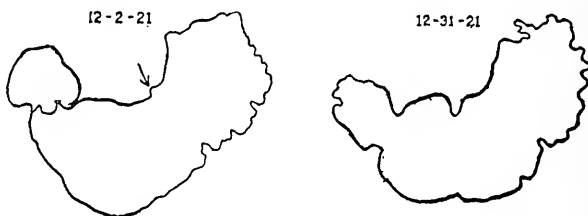
CASE A. R.

FIG. 5. December 23, 1921. Before treatment; big pouch at lesser curvature; duodenum fills very poorly.

FIG. 6. January 8, 1922. Immediately after treatment; the pouch is a trifle smaller; the duodenum fills much better.

FIG. 7. February 6, 1922. Prone; the pouch had disappeared.

FIG. 8. February 6, 1922. Same; erect.

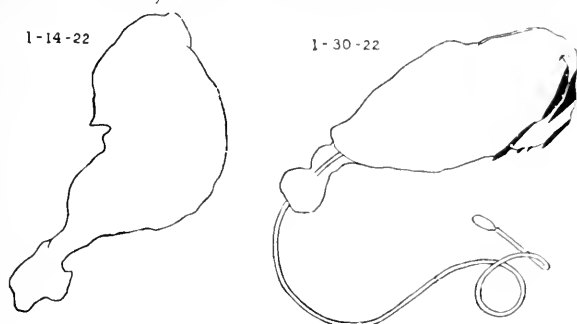


CASE E. K.

FIG. 9. December 2, 1921. Before treatment; small pouch visible at lesser curvature.

FIG. 10. December 31, 1921. After treatment; the pouch has disappeared.

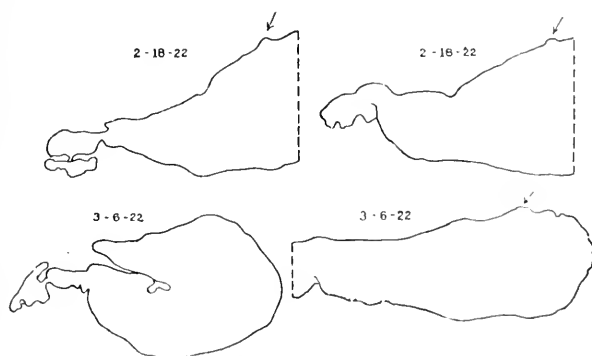
Since April, 1921, I have selected all cases of peptic ulcer with deformities (visible by X-rays) for reëxamination by the X-ray immediately after treatment, and in some of them some time later. In this



CASE S. D.

FIG. 11. January 14, 1922. Before treatment; small pouch at lesser curvature.

FIG. 12. January 30, 1922. Immediately after treatment; tube still in duodenum and jejunum; the pouch has disappeared.



CASE J. E. M.

FIG. 13. February 15, 1922. Before treatment; prone.

FIG. 14. February 15, 1922. Left side; pouch distinctly visible at the lesser curvature.

FIG. 15. March 6, 1922. After treatment.

FIG. 16. March 6, 1922. The pouch has almost completely disappeared.

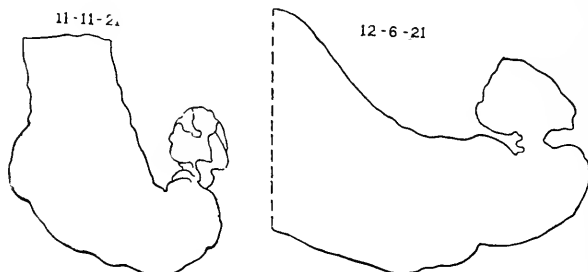
way it was possible to study with the eye the result of the treatment undertaken as influencing the pathological condition present. It is understood that not every case of peptic ulcer could be thus investigated, for many patients did not wish any X-ray examination or had



CASE H. A.

FIG. 17. January 26, 1922. Before treatment; small pouch or niche noticeable at pylorus.

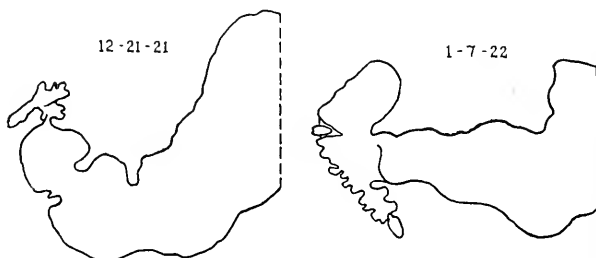
FIG. 18. After treatment: the pouch has disappeared.



CASE A. B.

FIG. 19. November 11, 1921. Before treatment; duodenal ulcer with pronounced deformity of cap.

FIG. 20. December 6, 1921. After treatment; cap almost normal.



CASE J. L.

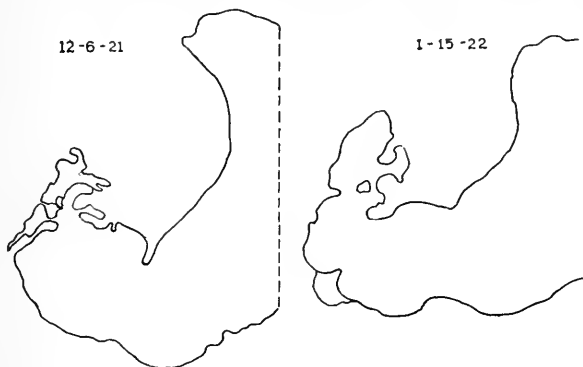
FIG. 21. December 21, 1921. Duodenal ulcer with marked deformity of cap before treatment.

FIG. 22. January 7, 1922. After treatment; the ascending portion of the duodenum appears normal and regular in outline.

it done some time previously. Or again, in some of the cases with deformities the patients decided to be treated surgically or by other physicians so that I had no chance to follow them up.

Altogether in this year (April 1, 1921, to April 1, 1922) 12 cases with deformities were treated by duodenal alimentation. A few of these I shall describe in detail while the rest will be given in table form:

*Case 1.* Louis K., April 3, 1921, aged thirty-seven years. The patient had been troubled for the past fifteen years with recurrent attacks of epigastric pains two to three hours after meals for periods of five to six weeks with free intermissions lasting from three to six months. Recently the attacks had become severer in



CASE S. H.

FIG. 23. December 6, 1921. Hodgkin's disease and duodenal ulcer. Before treatment; marked deformity of cap.

FIG. 24. January 15, 1922. After treatment; the deformity is considerably less pronounced, the beginning duodenum showing a better filling.

character, the pains being more intense and lasting longer. The patient also awakened during the night with pains, from which he found relief by eating crackers and milk. He became more restless and lost in weight and strength.

An X-ray picture taken at Philadelphia by Dr. Pfahler showed a penetrating (callous) ulcer of the lesser curvature of the stomach (fig. A). The patient was advised by several physicians to be operated upon, but a near relative of his having died soon after an abdominal operation the patient was loath to undergo the risk of the surgical procedure. Dr. Epstein, of Newark, N. J., a personal friend of the patient, consulted with me in regard to the best mode of procedure under these circumstances. We agreed to have the patient undergo a systematic examination and determine whether medical means would be applicable in this case. The gastric contents showed HCl, plus; acidity, 70; blood, negative; no food stagnation. The duodenal bucket string test showed the pylorus permeable; a blood stain at about 18½ to 19 inches from the lips, indicating the presence of an ulcer at the lesser curvature of the stomach.



FIG. A. Louis K., X-ray photograph taken April 1, 1921. A pouch of considerable size is visible at the lesser curvature. (Haudek's niche.)



FIG. B. Louis K., April 29, 1921. The pouch has diminished to about one-tenth its original size.

Inasmuch as I had seen several penetrating ulcers of the stomach apparently cured by duodenal feeding, I advised this mode of treatment before deciding upon an operation, which could be reserved in case of failure of the above mentioned procedure. The patient was then treated by duodenal alimentation for two weeks. He felt perfectly comfortable all the time, experiencing no pain from the time the duodenal feeding was instituted. Previous to withdrawing the tube I had new X-ray pictures taken by Dr. W. H. Stewart, one picture with stomach empty and another one after barium ingestion into the stomach. The latter photograph (fig. B) showed the pouch to have diminished to about one-tenth of its original size; two or three months later another X-ray was taken which demonstrated an almost entire disappearance of the afflicted area (fig. C). Immediately after the period of duodenal alimentation the patient picked up and could eat a



FIG. C. Louis K., June 16, 1921. The pouch no longer exists.

liberal diet with impunity. He improved steadily, gained in weight and strength, and remained well up to the time of this report. He could partake of banquets and could eat almost everything without restrictions.

*Case 2.* Andrew R., seventy years old, December 14, 1921. This patient complained of pain in the stomach three years ago, which usually came on about two hours after meals. Examination at that time revealed hyperacidity but no evidence of ulceration. Previous to this time the patient never complained of any stomach trouble. Since the patient was seen three years ago he had been enjoying good health until about two months ago, when he began to complain of severe pain in the abdomen. This pain was located in the epigastrium, would occur two to three hours after meals, and would spread over the entire abdomen.

At times he would vomit and this gave him some relief. The vomitus at times contained blood. At the onset of the present trouble food relieved the pain but lately it made it worse. Patient lost 20 pounds in two months, and when he



FIG. D. Andrew R., December 22, 1921. X-ray photograph shows a big pouch (Haudek's nicke) at the lesser curvature.

entered the hospital (Lexington Hospital) he was very weak. His bowels were constipated and he could not eat much. The pain was located in the epigastrium and radiated all over the abdomen.

Physical examination revealed an apparent tumor at the epigastrium. The man was much emaciated. A blood count showed the presence of a secondary anemia. The gastric contents showed HCl, plus; acidity, 45; occult blood present.

Duodenal string test showed a positive blood stain in the stomach. The stomach was dilated, the greater curvature extending to hands' width below the navel. On aspirating the stomach contents through the duodenal tube free blood was found. Wassermann reaction was negative.



FIG. E. Andrew R., January 8, 1922. The pouch appears a trifle smaller and the duodenum shows a better filling.

X-ray examination showed an extensive penetrating (callous) ulcer of the lesser curvature (fig. D). The patient absolutely refusing an operation, the duodenal tube was introduced, reaching the duodenum after three days' waiting and with the aid of frequent administrations of atropine. When the duodenal alimentation was established the patient began to feel better. After two weeks' feeding cautious alimentation by the mouth was substituted and the patient progressed rapidly (fig. E). Six weeks after the cure there was no trace left of the penetrating ulcer (fig. F). Patient had meanwhile gained fifteen pounds in weight.

*Case 3.* Harry A., fifty-one years old, December 5, 1921.

*Family history.* Mother died of cancer of the stomach.

*Past history.* For the past twenty-seven years the patient had had trouble with the stomach, i.e., dizziness, belching, headaches, and constipation. Four years ago he began to have pain two hours after meals. This pain would last a few months and then disappear for a month or two. The periodical attacks of pain lasted until six months ago when the pain was more or less constant after each meal.



FIG. F. Andrew R., February 6, 1922. The pouch has entirely disappeared.

*Present history.* For the past six months patient had a sharp pain in the epigastrium two hours after meals, which radiated around to the sides of the abdomen and usually lasted from an hour to an hour and a half unless relieved by taking food or bicarbonate of soda. Sour gaseous eructations followed the disappearance of the pain. Patient never vomited; was weak, constipated, but had not lost any weight.

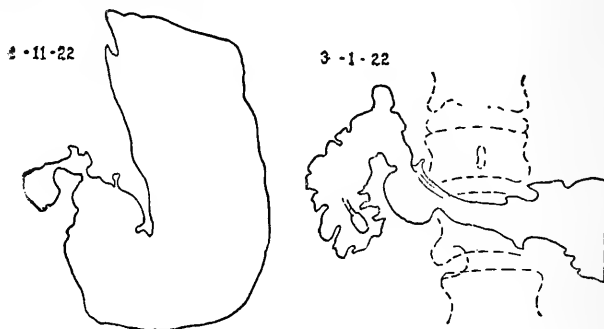
Physical examination showed a dilated stomach (splashing sound down to three fingers below the navel). Gastric contents: HCl, plus; acidity, 30; string

TABLE 1

| NAME                  | SYMPTOMS   | PHYSICAL SIGNS  | STRING TEST                                 | GASTRIC CONTENTS                                  | X-RAY   | RESULTS   |
|-----------------------|--|---|---|---|---|---|
| 1. E. K. ....         | Pain in epigastrium one hour after meals; lasts two hours; relieved by food and soda; no vomiting or hematemesis; tarry stools; ten pounds loss in weight  | Tenderness in upper part of epigastrium; stomach not dilated  | Positive; blood stain; 51 to 53 cm.         | HCl, 0; acidity, 0 blood and lactic acid negative | Penetrating ulcer of the lesser curvature                 | Ulcer and symptoms disappeared                                  |
| 2. S. D. ....         | Pain in epigastrium radiating to back two hours after meals and relieved by eating; no vomiting, tarry stools or loss of weight  | Tenderness in epigastrium   | Positive; blood stain; 47 to 48 cm.         | HCl, 10; acidity, 18; blood negative              | Penetrating ulcer of the lesser curvature near the cardia | Symptoms and ulcer disappeared                                  |
| 3. J. E. Mc. ....     | Gastric distress and hemorrhages from the stomach and the bowel (hematemesis and melena)   | Tenderness in abdomen; slightly enlarged liver  | Positive; blood stain; 48 to 52 cm.         | HCl, 50; acidity, 70; blood present               | Penetrating ulcer of the lesser curvature                 | General improvement; X-ray disappearance of the niche formation |
| 4. A. B. ....         | Pain in epigastrium ten minutes after meals; radiated to the left side and back; aggravated by food; regurgitation, sour material but no vomiting; tarry stools; good appetite; lost 11 pounds in weight | Tenderness in epigastrium, liver enlarged 4 cm. below costal margin                                     | Negative                                    | HCl, 39; acidity, 50; blood negative              | Ulcer near the pylorus with deformity                     | Symptoms disappeared; deformities in X-ray cleared up           |
| 5. S. H. ....         | Pain in epigastrium radiating to interscapular region; patient feels weak and lost 15 pounds in weight   | Tenderness in epigastrium; liver enlarged to five fingers below costal margin; enlarged inguinal glands | Negative                                    | HCl, 81; acidity, 99; blood negative              | Duodenal ulcer with gastric retention                     | Condition very much improved                                    |
| 6. J. L. ....         | Recurrent epigastric pain not related to meals; nausea and vomiting, relieved by food; eructations of gas; 17 pounds loss in weight  | Tenderness in abdomen   | HCl, 80; acidity, 100; blood present; trace | Positive; blood stain; 56 to 58 cm.               | Ulcer with marked deformity                               | Symptoms gone; X-ray shows great improvement                    |
| 7. A. G. ....         | Dizziness; epigastric pain, loss in weight and strength  | Dilated and prolapsed stomach; liver enlarged   | Positive; blood stain; 50 to 62 cm.         | HCl, +; acidity, 25                               | Duodenal ulcer with deformity of the cap                  | Greatly improved; deformity almost disappeared                  |
| 8. Mrs. C. H. R. .... | Pain in the epigastrium; loss in weight  | Dilated stomach   | Positive blood stain; 56 to 60 cm.          | HCl, 50; acidity, 70                              | Persistent defect in first portion of duodenum            | Defect greatly changed for the better                           |

test—blood stain at 55 to 56 cm.; bile below 60 cm.; X-ray revealed a niche right at the pylorus.

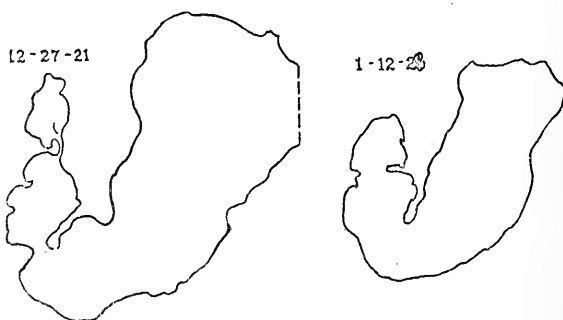
The patient was treated by duodenal alimentation for two weeks. An X-ray taken by Dr. H. A. Rafsky a few days after the duodenal feeding period revealed the disappearance of the niche and a normal cap. Patient now feels perfectly well and can eat a liberal diet.



CASE B. McC.

FIG. 25. February 11, 1922. Duodenal ulcer with marked deformity of cap (three leaf shape), before treatment.

FIG. 26. March 1, 1922. After treatment, the beginning duodenum, although not normal, shows a better filling capacity.



CASE A. G.

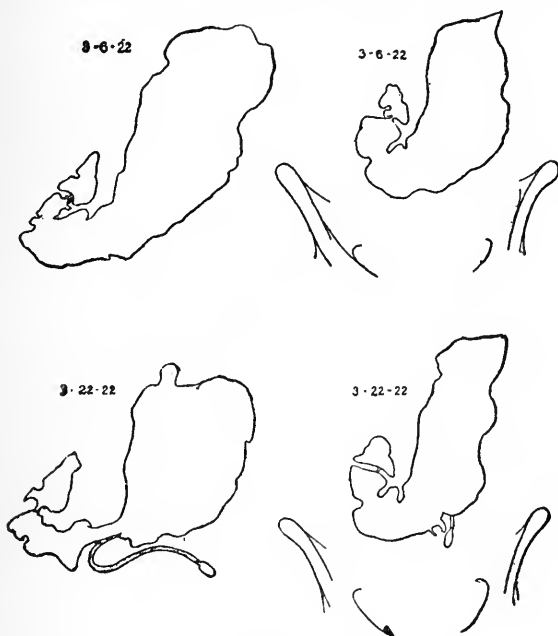
FIG. 27. December 27, 1921. Duodenal ulcer with deformity of cap; before treatment.

FIG. 28. January 12, 1922. After treatment; cap has an almost normal shape.

*Case 4.* Mrs. B. McC., fifty-eight years old, February 9, 1922. In August, 1920, while on a trip to Canada, the patient had an attack of nausea, vomiting, and diarrhea lasting two days. There was no pain but she felt very weak and had black stools; was jaundiced for two or three days with this attack but had not been since that time. Two to three months later she began to have a dull aching pain in the epigastrium which extended through to the back and had

become more frequent and severe. The pain had no relation to meals but was relieved by bicarbonate of soda; bowels were constipated; the patient had lost 10 pounds in weight.

Physical examination did not show anything of importance. Gastric contents, HCl, plus; acidity, 80; blood, negative; string test—blood stain 48 to 51 cm.; duodenal contents, light yellow, greenish tinge; turbid, alkalinity, 20; A, 7; S, 2; T, 3; few cholesterol crystals. The blood count showed a secondary anemia; the



CASE C. H. R.

FIGS. 29, 30. March 6, 1922. (Prone and erect); duodenal ulcer with filling defect in cap (slit); before treatment.

FIG. 31. March 22, 1922. After treatment; prone; slit still visible, though slightly.

FIG. 32. March 22, 1922. Erect; slit not visible.

urine, specific gravity, 1008; albumin, some white blood cells and many bacteria; stools were negative for blood. X-ray examination showed a persistent defect in the first portion of the duodenum (three leaf shaped cap) and six hour retention.

The patient was treated by duodenal alimentation for two weeks; no pains were felt since this régime was begun. At the end of the treatment a new X-ray was taken. The duodenum showed a much more normal course, the cap being present although not perfect in form. Patient is now well and is gaining in weight.

The remaining cases of peptic ulcer with deformities are given briefly in the table.

## REMARKS

In looking over the 12 described cases we find, as already mentioned above, that there were 5 penetrating (callous) ulcers of the lesser curvature of the stomach, 1 penetrating ulcer of the pylorus, and 6 clear cut cases of duodenal ulcer with constant deformities of the cap.

They were all benefited by the duodenal alimentation treatment; and there was a change for the better in the pathological findings as revealed by the X-rays, the niche formation having entirely or almost entirely disappeared and the cap deformities likewise changing to almost normal conditions. At the same time there was a general return of well being and health.

In one of these cases (A. R.) the change for the better in the niche immediately after the feeding was noticeable in a small degree, the niche becoming a trifle smaller and the duodenum filling up much better. The patient, however, continued to improve on a liberal diet and bismuth medication, so that in a period of about six weeks more, it had entirely disappeared, as the X-ray pictures well show (fig. F).

It is thus evident that even ulcers of long standing with marked pathological changes (or the so-called callous ulcers) of the stomach or duodenum are susceptible to cure by medical measures. Nor is this, according to my experience, a rare event. For the cases detailed have been taken seriatim and selected only for their gravity in demonstrating lesions easily recognizable by the roentgen method. (I here wish to express my thanks to Dr. W. H. Stewart, radiologist to the Lenox Hill Hospital, and Dr. H. A. Rafsky for the fine X-ray pictures taken for me.) As far as I recollect there was during the period (winter, 1921-1922) but one more case of penetrating ulcer of the stomach (visible by X-ray) in which I tried duodenal alimentation for four or five days but gave up the treatment on account of the persistence of pain and continued small hemorrhages. This patient was advised to be operated upon. Dr. F. Torek performed a gastro-enterostomy which cured the patient.

Based upon these experiences I must conclude that as a general rule most varieties of peptic ulcer, even the graver forms, are amenable to medical treatment. In my cases I have applied duodenal alimentation as a good method of supplying rest to the affected part and ample nutrition to the organism. But I do not doubt that similar results may be obtained by other methods of treatment, taking per-

haps a somewhat longer period of time to accomplish a cure. All these questions will of course require further study and elucidation.<sup>1</sup> But the main fact exists that peptic ulcers can be completely cured by medical measures.

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- (2) HAMBURGER, W. W.: *Amer. Jour. Med. Sci.*, February, 1918, 204.

#### DISCUSSION

DR. GEO. B. EUSTERMAN, Rochester, Minnesota: I have had considerable experience with both the usual and the penetrating form of ulcer of the stomach and duodenum during the medical treatment of over 200 cases in the past three years. Improvement in the luminal outline is the rule if the condition is not malignant. It is to be remembered that 10 per cent of all the deformities of the duodenum may be due to anatomical reasons or puckering of the mucous membrane with pocket formation as the result of complete healing. Another interesting observation is that today there may be a deformity of the cap, undoubtedly the result of spasm, which tomorrow might be absent, or vice versa. This obtains before treatment has been instituted and is known as spasm deformity. The effect of healing upon the luminal outline, especially in the duodenum, varies in different individuals. We have had repeated unique opportunity of noting the effect of healing when the stomach had to be reopened because of gastro-jejunal ulcer. Some duodenal ulcers heal out smoothly leaving little or no deformity on the pre-operative X-ray. Others will show extensive scarring and puckering, with resulting stenosis and deformity. While gastro-jejunal ulcer tends to reactivate the ulcer in many there is no sign of activity in the original lesion. We do know that chronic ulcers of the stomach and duodenum heal spontaneously whether they have given symptomatic evidence of their presence or not. We know this to be a fact as we frequently see it in operating on patients for other conditions or in the postmortem examination of patients dying from other diseases. In many patients successfully operated upon for chronic ulcer of the duodenum, who later come back for other conditions, we still see a marked deformity of the cap on gastric re-ray, in whom symptoms have been absent for many years. I have treated over 200 cases inten-

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<sup>1</sup> Since writing my paper, an article appeared from the pen of Dr. J. S. Diamond on "Observations on the curability of gastric ulcer, with report of fourteen cases of healed lesser curvature ulcers," in the *American Journal of the Medical Sciences*, April 19, 1922, p. 548. Diamond has observed and collected since 1916 14 cases with lesser curvature ulcers (niche of Haudek) which have been cured, symptoms having disappeared and likewise the niche. It took from six to eight weeks for the niche to disappear. Diamond has applied the usual modes of treatment of gastric ulcer, not taking recourse to duodenal alimentation. His results are in full accord with my own observations, and the medical treatment of peptic ulcer is thus enhanced in its value.

sively by the modified Sippy régime and I am skeptical about permanently curing the majority of them without a second course unless the coöperation has been very exact. I have found that the gastric deformity is more amenable to change following treatment than the deformity in the duodenum. In a few cases increase of deformity after healing provokes mechanical trouble which may require surgical interference.

DR. JULIUS FRIEDENWALD, Baltimore, Md.: Dr. Baetjer and I, I believe, were among the first to point out, in a paper read before the Association of American Physicians in 1912, the changes noted by means of the X-ray in the healing of gastric and duodenal ulcers. Since then, this work has been corroborated by Hamburger, in 1916, and more recently, by Ohnell, Buckstein and Diamond. We are still of the opinion, from examinations made long after treatment—in a large number of instances, even after years—that ulcers do heal, and that deformities that have previously existed disappear. The result of this work was interpreted by Dr. Baetjer, whose opinion, I believe Dr. Cole will admit, is worthy of the most earnest consideration.

DR. CHARLES G. STOCKTON, Buffalo, N. Y.: Ulcers heal, and remain healed for years or indefinitely. We are making examinations after years, to see whether any trace of old ulcers can be found. I think that in no field have I seen such strikingly beneficial results from duodenal feeding as in ulcer. Even if seated on the lesser curvature, ulcer will heal. I have seen cases corresponding with those that Dr. Einhorn has presented, and have not only X-ray evidence of recovery, but what I consider more emphatic proof, the clinical history extending over many years. Some of these cases were very stormy in the beginning, succeeded by striking relief. I do not believe the value of duodenal feeding in ulcer on the lesser curvature is a matter sufficiently appreciated.

DR. JACOB KAUFMANN, New York City: I want to make only one remark, and that is this: While it is true that the disappearance of the deformity may be taken as proof of the healing of the ulcer, the fact that deformity remains is no proof that the ulcer has not healed. We are not treating the anatomical defect but the disorders of function, which produce the defect. We often find at postmortem defects from very large ulcers which are perfectly healed. What we are treating is the ulcerative process. That is the thing which we are aiming at. The healing of the ulcerative process and the preventing of recurrence of the ulcerative process is possible, even when a marked defect remains eventually evident on roentgenologic examination.

DR. WILLY MEYER, New York City: To my mind the observations made by Dr. Einhorn are of great clinical importance. They prove the narrowness of the border line between medicine and surgery in these cases.

Up to the present time patients with gastric ulcer which the X-rays has shown to be penetrating, have been referred to the surgeon for operation. Now Dr. Einhorn proves by his radiograms taken before and after treatment with the duodenal tube, that some of these cases at least, can be improved if not cured without operation. The typical niche, present before treatment in every picture, has disappeared in

the pictures taken after treatment; the lesser curvature of the stomach presents an unbroken line.

Even granting Dr. Cole to be correct in throwing out 9 of Dr. Einhorn's 12 films, the fact then remains that Dr. Einhorn has shown 3 cases of undoubted, penetrating gastric ulcer, the clinical symptoms corroborating the radiographic findings, in which he succeeded in clinically curing the patient, temporarily at least, by means of duodenal feeding.

Of course, such experience does not at once definitely change the indication generally accepted so far. All these cases will not now remain in the hands of the internist. Still, in the event of a patient's objecting to operation, or complications forbidding it, the advice to try duodenal feeding would seem well founded.

It certainly is remarkable that, as Dr. Einhorn repeatedly observed, definite improvement set in after two weeks' treatment, although the rich blood supply derived from four powerful arteries easily explains this. Personally, I would feel inclined to put the stomach at complete rest for at least three weeks.

Of course, a careful following-up of these cases is a *sine qua non*, for the question to be determined is: will the improvement or cure be permanent, or temporary only.

The observation made by Dr. Einhorn in the case of the doctor of medicine would seem to show that the duodenal tube treatment *can* bring a real and permanent cure in these cases.

DR. LEWIS G. COLE, New York City: In some ways, I have had an advantage in that I have been able to have carefully studied the paper that Dr. Einhorn has presented, as it was submitted to me as a member of an editorial committee. I must confess that I have studied it with very great care, have made a few notes here, and should like to read them.

The field which Dr. Einhorn has scratched over is certainly a fertile one, and one which deserves real thorough cultivation; and his method in using a series of roentgen examinations as a proof of the cure is an excellent one, and one which we have referred to in the past as the natural history of disease.

Dr. Einhorn has stepped out into the arena with a big lance, and has challenged Sir Berkeley without having a horse to ride, except a hobby, and without an armor to protect him from the thrusts of Sir Berkeley and his admirers.

Dr. Einhorn says surgeons, physicians and radiographers would all unite in making a positive diagnosis of advanced ulcers in these 12 cases, and intimates that they would agree concerning the X-ray evidences of a cure.

So far as you surgeons and physicians are concerned, you must speak for yourselves; and so far as the radiographer is concerned, he may be right: but what is a radiographer. He is a technician, or a non-medical man or woman, experienced only in the making of radiograms, and without any knowledge of medicine; and usually in the employ of a doctor and, as such, is wise if he concurs in the opinions which are of greatest advantage to his employer; and if Dr. Einhorn had consulted a competent roentgenologist (a physician qualified in the interpretation of roentgenograms), he would not have so boldly challenged Sir Berkeley.

All of us who know Dr. Einhorn so well, respect him for the vast amount of work that he has accomplished, for his zeal in trying new untried methods in diagnosis and treatment, and for the promptness with which he reports his conclusions. In the

method of clinical and laboratory diagnosis with which he is familiar, every one respects his opinion; but for so many years he was loath to employ the X-ray in diagnosis that he did not learn to interpret roentgenograms during the development of the speciality—and now, at this late date, he attempts to employ it in the most advanced and difficult of all roentgenological work, viz., in determining whether or not ulcers, particularly postpyloric ulcers, have healed—or, rather, he employs it to prove that under duodenal alimentation, gastric and duodenal ulcers “change for the better” (a very safe and elusive statement).

A very careful study of the prints and drawings of the 12 cases submitted in proof of his contention, that as a result of duodenal alimentation ulcers have “changed for the better,” leads me to believe that in only 2, or possibly 3, of the 12 cases does the roentgen evidence warrant his conclusion.

These are the first 2 cases that he reports; and if he had rested his case on the evidence of these, he certainly would have had Sir Berkeley on the defensive.

But not in any of the roentgenograms of the remaining 9 or 10 cases made before and after taking the tube, does the roentgen evidence prove that they are “changed for the better.”

A rapid analysis of these 12 cases, made after a thorough study of the evidence submitted, leads to the following conclusions:

Case 1: The roentgen evidence proves the healing of the ulcers.

Case 3: The crater indicated may be an ulcer, but I believe that it is more likely a peristaltic wave; as there is no evidence of lack of pliability of this repair in the plate made after the cure.

Case 4: The deformity indicated as a crater is due to a ruga coming to the surface of the opaque meal, and is just as evident in the plate after as before.

Case 5: The crater is probably the cardiac orifice, and is entirely obscured by the position of the stomach in the plate made after taking the tube.

Case 6: Is a crater on the anterior surface of the cap and the cap is in a state of spasm; and in the plates made after the cure, the crater is partly obscured by the filling of the uninvolved portion of the cap.

Case 7: Shows the typical deformity of a postpyloric ulcer, without evidence of a crater; and in the subsequent plate, this is partly obscured by dilation of the upper part of the descending duodenum.

Case 8: Cap deformity without X-ray evidence of crater, and with spasm; after treatment, the cap deformity without spasm.

Case 9: Radiograms, both before and after, show the crater; but the first plate is lateral or oblique, and is compared with an anterior plate.

Case 10: No crater shown in first radiogram, cap and descending duodenum give a shadow which is misinterpreted as a normal cap.

Case 11: Shows a fold or crease, indenting the cap, interpreted as a crater; and the radiogram made after treatment shows almost exactly the same indentation in the cap.

Case 12: Shows slight deformity in the cap, no crater, with the patient prone; and this is probably due to pressure of the descending duodenum on the right side of the cap, and possibly to slight adhesions. After treatment, the radiogram was made with the patient in the erect posture; but practically the same deformity is shown.

In conclusion, I would state that I have urged these repeated X-ray examinations, to determine whether an ulcer has healed under either medical or surgical treatment; and the surgeon has no more right to claim a cure than has the medical man. I have referred to the subsequent roentgen examinations as the "acid test" to which all ulcer cures should be submitted; but to be of value, the roentgenogram must be interpreted by a competent roentgenologist, and not by a radiographer or technician; and under no circumstances should the defendant be his own judge and jury.

DR. MAX EINHORN, New York City (closing): I have not much to add. I would only say that the pictures were taken in the Lenox Hill Hospital, New York. I always asked the gentlemen who took the pictures after treatments to have the same positions as when the pictures were taken before treatment, so that we could compare them. There may be one or two that are not so very characteristic, where the cap has not entirely re-formed to its normal shape; but that does not change my paper. What I wanted to bring out is that in many cases we can cure ulcer of the stomach or duodenum. That was shown not only by X-ray, but by the well being of the patient, and so on. There will be cases in which it may be doubted whether there is anything malignant present. I always say, when we are in doubt, that we should then consider operative intervention; but I collected those cases in which, by all the means at our disposal, we could make a diagnosis of a benign condition, and in which this method could be very well tried and without much fear; and we have been very successful.

## VEILS IN THE RIGHT HYPOCHONDRIUM

LEWIS G. COLE

*New York, N. Y.*

Veils or membranes or slight non-obstructive adhesions in the right hypochondrium, especially involving the gall-bladder and its ducts and the adjacent hollow viscera are of relatively frequent occurrence, in fact they are of such frequent occurrence that one may ask "Are they of any significance?"

This question must be considered from three aspects: (1) the roentgenological; (2) the clinical, and (3) the surgical.

1. From an X-ray standpoint, they are of great significance, because the various types of veils cause gastric and cap deformities that are so similar to those of gastric cancer or ulcer and especially so similar to post-pyloric ulcer and gall-bladder adhesions, that most of the erroneous X-ray diagnosis that occurs when a sufficiently extensive series of radiograms have been studied by a competent observer are due to these veils or membranes. The roentgenological aspect of this subject has recently been considered by the speaker in an article read before the American Roentgen Ray Society and published in the March issue of the American Journal of Roentgenology. At that time the X-ray findings were described and therefore they will be eliminated from this article, except as they will be demonstrated on the slides.

The clinical significance of veils in the right hypochondrium: From the agitation that has resulted in medical and surgical circles because of similar veils in other quadrants of the abdomen, I am loath to add fuel to the fire of discussion concerning Lane's kinks and Jackson's membranes, which, at the present time, seems to be smouldering, or at least their significance is relegated to a position of second or third magnitude, compared with a group of thoroughly recognized lesions such as cancers, ulcers and grossly involved pathological gall-bladders, which are stars of the first magnitude in the heavens of every surgeon.

Morris (A. Med., July 15, 1905) very picturesquely described the veils as "cobwebs in the attic" of the abdomen and considered them

of "gall spider" origin and of clinical significance at least in some cases. He stated that "when there is an infection on the mucosal side of these thin walled structures (the gall-bladder and its ducts), toxins penetrate the walls in sufficient force to cause a desquamation of the endothelium on the peritoneal side, endothelial cells are shed, plastic lymph exudes, coagulates and is replaced by connective tissue, and "webs of adhesions are spun" from the biliary tract to the adjacent hollow and solid viscera. Harris (Jour. Amer. Med. Assoc., April 1, 1914) describes veils in the right hypochondrium and at first he too considered them of gall-bladder origin, but subsequently from a study of 6 operated cases and a fairly extensive study of the embryological development of this region he thought that they were abnormal folds in the anterior mesogastrium. He, too, considered that they were of clinical significance and demanded surgical treatment.

I believe that both Morris and Harris were right. Some of the veils are of "gall spider origin" and others are the result of abnormal formations of the anterior mesogastrium, and in many instances one can determine from a serial roentgenological examination which of these conditions cause the veil and I believe that both men were right in considering that they cause symptoms, but I must emphasize the fact that they are not of the same magnitude as cancers or ulcers.

These veils are often associated with symptoms that are out of proportion to the extent of the pathology and again we may find characteristic veils with very few symptoms.

The symptoms often closely resemble those of post-pyloric ulcer, except that the time interval is not so characteristic and they are usually worse when the patient is standing or riding in an automobile and there is not the night aggravation which is characteristic of post-pyloric ulcer, and the pain is often relieved by lying down.

In other cases the symptoms resemble those of a chronic gall-bladder infection, with a dragging or boring pain often referred to the back or right shoulder, and with the ever present symptom which the patient refers to as gas in the stomach. They do not have the acute attacks of biliary colic nor the temperature of an acute or a sub-acute gall-bladder infection. If the veil involves the hepatic flexure the symptoms may be aggravated by constipation or encarceration of gas in the hepatic flexure.

Veils in the right hypochondrium are of interest from a surgical standpoint—first because of the question of whether they demand

surgical treatment, and, second, because they may be overlooked by the surgeon.

The question of whether these veils demand surgical treatment depends on the severity of the symptoms and whether the symptoms are characteristically those of the veil.

In 1 of the cases which will be shown on the slides there were severe periodical attacks of pain referred to the region of the umbilicus with temperature; a complete series of radiograms failed to show anything except a rather characteristic veil and this was described in the roentgenological report, but I stated in the diagnosis that I did not believe it caused the symptoms of which the patient complained. The symptoms persisted and an exploratory laparotomy was done and the veil was demonstrated and severed, but the symptoms persisted the same after the operation as before.

The simple X-ray demonstration of a veil, no matter how characteristic, is not sufficient evidence on which to advise surgical treatment unless the symptoms are characteristically those of a veil and then only when the symptoms are severe and have been prolonged, and under no circumstances should the X-ray findings be used as an excuse for surgical procedure for the relief of indefinite symptoms of which many hypochondriacs complain.

On the other hand if the patient complains of symptoms sufficiently severe to demand surgical procedure and the X-ray shows a deformity of the cap so constant as to be diagnosed as an ulcer and if on surgical exploration no ulcer is found either on the anterior or posterior surface of the cap, then the surgeon should search diligently for the cause of the cap deformity and in many instances it will be found to be due to a veil or membrane of the types about to be shown, and if this veil is found and severed, the roentgenologist will be partly relieved of the humility of an erroneous diagnosis, the surgeon will have been justified in opening the belly and the patient will probably be relieved of the symptoms of which he has complained.

#### DISCUSSION

DR. MAX EINHORN, New York City: I had the opportunity to see a patient, about six months ago, who came to me with a diagnosis of duodenal ulcer. X-rays were taken, I think twice, by a specialist; and this diagnosis was made. The patient was in the hospital under my care. I made all the tests, and could not find any evidence of high acidity; but I found, on examining the gall-bladder contents in the fasting condition, an acid bile, very turbid, showing that he had some

gall-bladder trouble that should be treated. An operation was done by Dr. William Mayo; and I had the opportunity to inspect the duodenum, and also the gall-bladder, at the operation. No trace of an ulcer was found, but adhesions of the gall-bladder and the duodenum were discovered. These were very similar to what Dr. Cole has illustrated in these photographs. The gall-bladder was resected. There were no stones. Recovery took place. I want to mention that deformity of the cap alone is not sufficient to make a diagnosis of ulcer of the duodenum on, as Dr. Cole says.

## THE COLON

MARTIN E. REHFUSS

*Philadelphia. Pennsylvania*

The colon as a factor in various diseased conditions has received at times the most extravagant recognition. On the other hand there has been a tendency on the part of the profession to ignore its importance, and to meet every anomaly of colon action with the exhibition of laxatives or cathartics which are supposed to be a cure-all for any condition which might supervene. To give a laxative in an attempt to cure a colon condition is about as sensible as to expect a chronically diseased tonsil to yield to a single application of an antiseptic; the logic is the same, so are the results. But throughout the world today, and particularly in France, there is a revival of interest in the question of diseases of the colon. This interest is a very different one from the extreme views which have been enunciated from time to time which designate the colon as a factor in the determination of every known condition. The present day studies, as shown in the literature, are very real attempts to determine the status and pathological significance of the colon.

An intelligent survey of the digestive tract reveals the colon as an organ which has differentiated for its own special function, namely, the absorption of water, the final digestion (principally bacterial) of cellulose, and eventually and most important, the function of elimination. The colon has no great surface area (intensified by the valvular conniventes and villi) as has the small bowel, nor has it the rapid peristalsis which the small bowel demonstrates. On the other hand its movements, with the exception of the mass action of the colon, are essentially slow, vermicular, and plastic rather than disintegrative; and the sum total of its work is to concentrate, reduce and eliminate the final products of digestive work. Probably the most important single factor in this country, today, is the incidence of incomplete elimination on the part of the colon, or what has been styled fragmentary constipation. Constipation is sufficiently common, but even more frequent is the passage of movements which are

insufficient and which constitute one of the several forms of failure in elimination.

It has long been known that there is a very real difference in the clinical effects of intestinal stasis, depending on the level of stasis. Stasis in the sigmoid can be endured for long periods without any untoward effect; stasis high up in the colon (in the cecum and ascending colon where the contents are liquid and bacterial proliferation marked) can be endured only for short periods without the intervention of clinical symptoms.

If stasis is associated with the proliferation of pathogenic organisms but one result can follow, namely, infection; and even in the absence of pathogenic organisms, sooner or later, infection. The determination of infection at the head of the bowel is still a clinical point which as yet has not been completely solved. That stasis in this portion of the bowel is in a sense responsible for the lack of resistance to infection elsewhere in the body is evident from a large series of cases which I have collected, in which the incidence of colon bacilluria, infection of the biliary tract and the association of upper respiratory infection is abnormally large. Furthermore, the disappearance of these foci is at times extremely difficult in the face of a demonstrable and persistent stasis of the colon. I have made, with the assistance of Dr. E. D. Funk and Dr. John Kolmer, a bacteriological study of these findings in several hundred cases of colon stasis. The conclusion of these studies brings out the fact forcibly that colon stasis unquestionably reduces body immunity to focal infection.

We have at our disposal the following means of examining colon disease: (1) physical examination, (2) X-ray examination, (3) sigmoidoscopy, (4) examination of the feces, and (5) recurrent irrigation. It is the last method in particular that I will discuss in this paper, but it is a discussion based on a method of approach. Before this subject is resumed I would like to briefly discuss the other headings.

#### PHYSICAL EXAMINATION

Physical examination of the colon is of value in proportion as the fingers of the examiner are trained and the physical condition of the subject facilitates the study. In a thin subject, with a flaccid abdomen, it is frequently possible to palpate almost the entire colon. In the average case it is possible to palpate the cecum, the ascending

colon on the right side and descending colon on the left side. I find the method of palpation with the hand placed almost flatly on the abdomen (not with the tips but the pulps of the fingers) the most valuable procedure. If the observer uses a gentle, sliding movement up and down the axis of the colon, and again a horizontal sliding movement from the umbilicus to the iliac crest, it is usually possible to slide the cecum and ascending colon under the fingers and feel its resiliency, its degree of repletion and mobility, as well as the extent of tenderness associated with such a procedure. Normally the material in the cecum and ascending colon is liquid, and it is possible to palpate deep in the iliac fossa and displace the soft resilient head of the bowel with a slight gurgling sound. This portion of the bowel, however, should never contain solid material; and it is precisely those types of high ascendens constipation, with solid material retained in the head of the bowel, that I wish to discuss. In my experience there are two types of solid ascendens; the palpable cecum, and the ascending colon with dough-like fecal accumulation. One is the type with little or no tenderness which is seen in atonic dilatation of the cecum and ascending bowel; the other is the tender type of the same phenomenon with diffuse right-sided tenderness, often extending up to the hepatic flexure and obviously associated with manipulation of the bowel. This type is usually associated with tenderness over the descending colon and the tender "corde colique" of the French. The one type is clearly an arrest in an inflamed, spastic bowel. Both are frequently mistaken for chronic appendicitis, and in my experience in both cases (but particularly in the second variety) there is tenderness over McBurney's point. The appendix may or may not be inflamed, but the fact remains that the removal of the appendix does not relieve the case and colon phenomena are as persistent as they were in the beginning. These are associated with pericolitis, colitis and typhilitis. This does not conflict in the least with the fact that in many of these cases a defectively acting and inflamed appendix needed removal. I merely want to emphasize the fact that the appendix is but 3 inches of the 50 or more inches of inflamed bowel.

#### X-RAY EXAMINATION

X-ray examination more than anything else has helped to define the status of the colon. From X-ray studies we have defined the anomalies in colon position; diagnosed adhesion formation, fixation

and ileal regurgitation; recognized ptosis, redundant sigmoid and a whole series of ptosed conditions—such as a prolapse of the cecum into the pelvis, a general right-sided position from faulty congenital, rotation, etc. Concerning colon position the consensus of opinion as far as I can gauge, is very similar to that of the stomach, and visceral position is far less important than visceral function. Efficient function is the paramount point, and only when faulty position interferes with efficient function does it assume importance. I do not wish to be credited with the statement that faulty position is not in itself evidence of systemic trouble, or that faulty position in itself is not responsible for many symptoms. On the contrary I recognize clearly that both of these are common associations. What I do want to make clear is the fact that many low bowels as well as many low stomachs continue to operate efficiently until something *happens*; and function suffers, and with an interference with function a vicious cycle is instituted. What that something is, is beyond the province of this paper; the important point is that a disturbance in function is the sequence, and in the case of the colon a disturbance in elimination with its resultant vicious cycle. It seems to be accepted clinical fact that the higher up the stasis the more dangerous it becomes. Esophageal stasis needs no mention for gastric delay soon makes itself felt. Small intestinal delays are apparent, but a low grade chronic obstruction (which is precisely what every form of intestinal stasis resolves itself into) produces only subacute but nevertheless insidious inroads into the general economy.

I have personally fluoroscoped the bowel over two thousand times in the last few years. In 348 cases of pronounced constipation 136 or 39 per cent showed all the evidence of atonic constipation and there was no area which indicated spasm. In 131 or 37 per cent of cases there was definite evidence of spasm, narrowing of the lumen, mucous in the movement and high retention. In 39 or 11 per cent of these cases the appendix had been removed, and in over one-half a chronic appendix supposed to have been the cause of the trouble was removed without eventual relief. In 136 or 39 per cent of cases there was evidence of cecal and ascendens retention. In 73 or 20 per cent there was evidence of cecal prolapse downward toward and in the pelvis. In 37 or 10 per cent of cases there was clean cut cecal and ascendens dilatation, and in 14 or 3.1 per cent evidence of inversion and delayed descent of cecum or reduplication. In my judgment only 7 or 2.3 per cent of cases belong to the dyschezic type. In 24 or 6.9 per cent

the transverse colon lay on the pubic bone in the upright position, and fully three-fourths were below the umbilicus. Marked sigmoid redundancy was seen in 12 cases; 21 or 6 per cent showed distinct adhesion formation, 5 to the gall-bladder, 11 from the appendiceal region, 2 between the ascendens and the transverse, 1 between the stomach and transverse colon and 2 to the pelvic viscera. The ratio between the diameter of the ascendens and descendens as noted in this number was 1.5:1 in 44.8 per cent or 156 cases, and 2.1 or over in 135 or 38 per cent of cases.

#### ANALYSIS OF COLON CONDITIONS BY FRACTIONAL IRRIGATION

Irrigation of the colon is a procedure which has been known for a long time and which has been carried out in many different ways. From time immemorial the enema, and later an actual irrigation, together with even the complicated methods (such as irrigation under pressure) have been devised for colon complaints. There is probably no procedure in medicine which is performed so differently, or regarding which so many divergent views have been held. Its therapeutic value can be reserved for further discussion; the point which I want to emphasize for the present is the diagnostic value of this procedure, the method of performing it and the facts to be ascertained by its use.

For this purpose I simply use the Y tube, with a large soft stomach tube as the rectal tube and soft tubing of a caliber as large or larger than the glass Y. In the efferent or lower tube I use a glass T tube with a small piece of tubing and pinchcock, which can be inserted to draw off specimens for examination as they are desired. By this simple expedient it is possible to obtain specimens from different segments of the bowel and examine them grossly or microscopically. For seven years I have used this procedure with my bowel cases, combining irrigation with manual expression on the abdomen. The rectal tube is inserted into the rectum about 3 inches, never over 4 inches. Tube B connects with the pail, or a large container, of at least 1 gallon capacity. Tube C, which is the efferent tube, goes to a pail on the floor, and the side arm D is used when it is desirable to draw off specimens. The part connected to tube B should never be higher than 4 feet (and preferably 3 feet) above the level of the patient. Our rules regarding this procedure are as follows:

(1) Plain syphon water is used. It should be somewhat above blood heat, but we have never found it necessary to use a thermometer.

(2) There are on both B and C stopcocks which are shut off when it is desirable to use the hands for some other purpose.

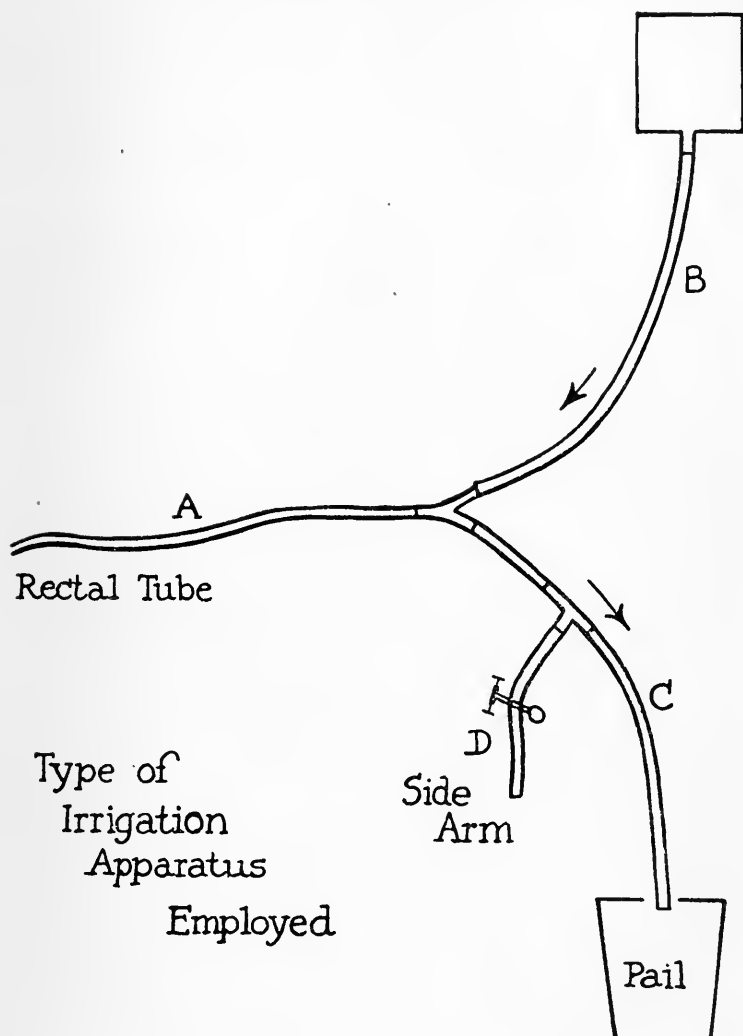


FIG. 1.

(3) The irrigation is performed by exerting alternating pressure on tube B and C. Ordinarily the nurse sits on a chair, seizes tube B in the right hand and tube C in the left, and grasps the tube in such

a way as to exert pressure between the thumb and index finger. In this way it is possible to *feel* not only the current of water, but also the arrest of the current through blockage of the bowel or of the tube.

(4) Only about 4 ounces of liquid is introduced at one time. This is a most important point, inasmuch as the common habit of pupil nurses is to fill the bowel; a point which defeats the primary purpose which is irrigation of the colon.

(5) After every introduction tube B is closed and tube C allowed to remain open, so that there is never overdistention of the bowel. This is a point which many nurses fail to grasp.

(6) Note is made of the condition of the abdomen. This is the point which only the experienced physician can teach a nurse by repeated practice. A bowel, particularly one with retention, is readily felt by skilled hands on manual palpation. The nurse who is trained in the work is cognizant of this fact, and she knows when she has emptied the bowel. There is a total disappearance of the sense of resistance either in the left or right iliac fossa. But to produce this total disappearance of the dough-like resistance of the right iliac is the work of one to two hours and often many gallons of water. It frequently takes 5 to 6 pails of water to produce a condition of the colon which physical examination demonstrates to be that of an empty bowel. I have on innumerable occasions examined individuals who have daily movements, or after profuse cathartics or even diarrhea, and found indisputable evidence of marked retention which is convincingly demonstrated by colon irrigation. If one bears in mind the fact that there should never be solid material in the right iliac fossa but simply a soft gurgle, it is not difficult to realize the other condition which is a most common one. I have no hesitancy in saying that neither enemas nor cathartics can or do correct a condition of this kind, and when pronounced palpable stasis of the right colon results the only method which is productive of results is a mechanical one, such as irrigation.

From a diagnostic angle this procedure presents two possibilities: (1) the way in which the bowel reacts to this method, and (2) the examination of the specimens removed at intervals.

#### THE BEHAVIOR OF THE BOWEL TO IRRIGATION

1. Immediately after the institution of irrigation there is a desire of the patient to go to the stool, or there is an immediate evacuation of material from the efferent tube. This is the typical

type of response found in rectal constipation with retention, and is usually followed by the evacuation of the bulk of the material immediately or in the first twenty minutes. This material is usually well digested and indicates from its appearance the long duration of material in the bowel.

2. The second possibility is thorough and persistent irrigation, with material which continues to stain, discolor or fill the wash water from the inception of the irrigation, throughout the irrigation to the end. This type is encountered in those forms of atonic constipation which line the entire bowel.

3. The third type is that in which there is no result from irrigation for one-half to three-quarters of an hour, and then almost as if a mine was tapped the material begins to flow. It is this form which is the common form and in which the retention is almost all high colonic. The appearance of the material is coincident with the slow disappearance of the palpable mass at the head of the bowel. This may or may not be followed by a movement after the irrigation.

4. Finally there is a form in which irrigation fails to produce any results, but following which a movement occurs undeniably induced by the procedure and almost always coming from the head of the bowel.

As variation of these forms are the following possibilities:

1. Irrigation absolutely fails. It is next to impossible to irrigate and an attempt to continue the procedure only results in pain to the patient. This is the form with spasm, usually of the sigmoid. In severe spasm of the left colon the nurse will know that such is the case, inasmuch as it is impossible to introduce much material in the bowel. It is interesting to observe that in many instances, if the procedure is persisted in, the left side of the bowel relaxes and the method can then followed without interruption.

2. The irrigation brings down only mucus, and mucus throughout the irrigation naturally speaks for a colitis; mucus in the first portion for an inflammation low down; and mucus towards the end of the irrigation for inflammation high up in the colon.

The combined observance of palpatory and irrigation findings yield information of the greatest interest. The appearance of fecal material, with a disappearance of a resistance in the line of the colon, would argue for the point of resistance as the source of the material.

Many points are noted in carrying out this method: the nature and character of the material; the presence of undigested food; the presence of mucus, pus or blood, and the color and odor—all of which argue for certain conditions in the bowel. The slimy, fetid material found in certain forms of colitis, and the material from intestinal fermentation cases, are highly characteristic. On the other hand an examination of the material when drawing it off, and its examination microscopically, yield many points of the greatest interest. In a normal individual it is possible to obtain material from the bowel but the material is very different from that of actual and marked stasis. That the cecum is in truth the organ for cellulose digestion is apparent by the richness of cellulose which is found in the final specimens. In true stasis the material from the head of the colon approaches the type seen in the descendens and sigmoid.

After irrigation the following phenomena may occur:

1. The absorption of water often induces marked diuresis and even sweating.
2. The patient may be exhausted but will feel much better later on in the day or the following day, a phenomenon which I would ascribe as similar to the negative and positive stages of a vaccine reaction.
3. The patient has an immediate and very marked sense of well being, obviously due to the removal of toxic material.
4. The patient may have considerable abdominal discomfort or even localized soreness; a finding which in the light of a large experience with this procedure usually indicates some inflammatory focus in the abdomen.

Inasmuch as the overwhelming majority of responses fall in the first three groups, it follows that in the first three groups are to be found those who derive benefit from the procedure.

Another point which is worthy of consideration is the aftermath of these cases after irrigation: (1) some are permanently relieved; (2) a second group, and by far the majority, slowly fill up in the same place; (3) only those are made worse which have phenomena of inflammation.

It is evident, therefore, that irrigation properly carried out enables us not only to determine the extent and character of stasis in a very direct sort of way, but it gives us information of the position of stasis. Inflammation is naturally determined by the presence of mucus, pus and blood. Obviously the combined fecal study, colon

fluoroscopy, irrigation, physical examination and sigmoidoscopy are the procedures which the clinician can then use in studying colon disease. The bacteriological examination of the stool will be the procedure which will become more and more routine as its rationale is understood. Personally we are content to examine the flora rou-

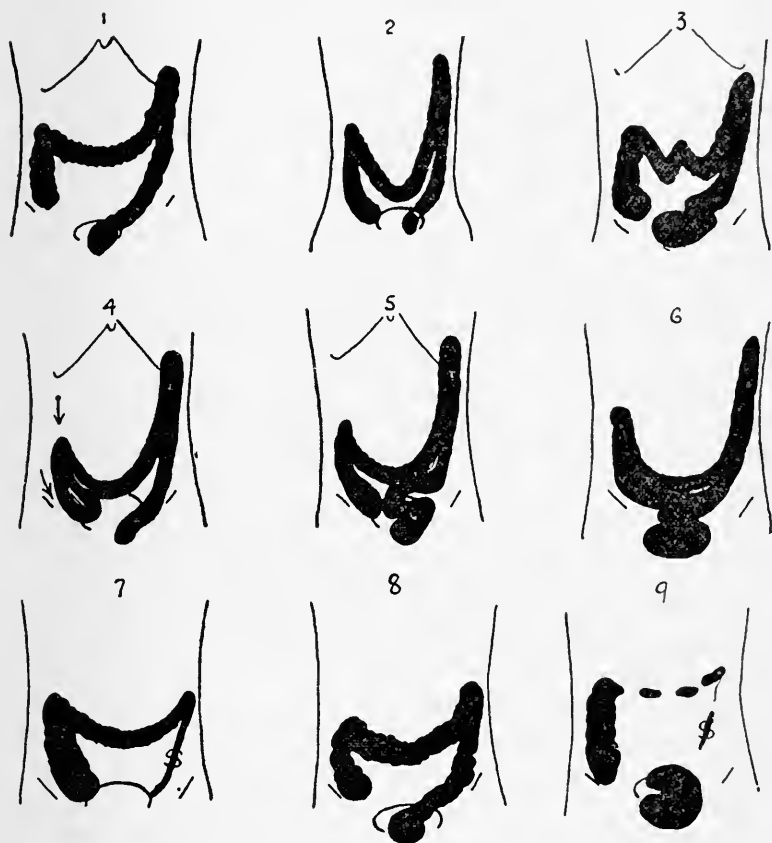


FIG. 2.

tinely by Gram stains, sending only those cases to the bacteriologist in which there is evidence of the preponderance of certain pathogenic bacteria.

The common picture of the colon (type 1) on the X-ray screen reveals a bowel which varies in form and position according to the habitus and state of nutrition of the patient. Type 2 is the common form encountered in the long, slender individual congenitally pre-

disposed to visceroptosis. It may be argued that this is not a normal type; on the other hand there is undeniable evidence that this type does perform its function as efficiently as does the other type. I believe, however, that type 2 is predisposed to bowel disturbance far more frequently than type 1, owing to the inherent instability and weakness of both the muscular and nervous mechanism. In type 3 is to be found a distinct group of M colons, and also a tendency toward cecal prolapse on the right side and redundant sigmoid on the left.

As variations in the bowel probably the most common variation is type 4 in which there is a prolapse downward of the right side of the bowel into the pelvis; with a descent of the cecum, ascending colon and hepatic flexure. This is the type so eloquently discussed by Lane and producing in its descent Lane's Kink, and this is likewise the type in which the high colon stasis is found. Type 5 represent those cases in which there is redundancy of the sigmoid and left side of the colon of varying degrees, all the way from slight redundancy to severe cases in which the tremendously redundant colon goes over to the right side. In type 6 is diagrammatically pictured advanced ptosis with an almost complete prolapse of the right side, a transverse colon on the pubic bone, and even a superimposing of the sigmoid flexure on the rectum. In thirty-six and forty-six hours the type of constipation becomes manifest and it is evident that they are usually of three types. Type 7 is the ascendens type; most pernicious in its effects, and if persistent and long standing almost always engendering upper abdominal trouble. Type 8 is that with uniform stasis throughout the colon which is best seen in the atonic cases, and type 9 is the rectal or "dyschezic" type of Hertz.

These forms are readily recognized from every day pictures in the study of large intestinal disturbances. The picture of chronic appendicitis is of course a varying one; the fixation of the cecum, the visualized appendix which is pushed or tied in some portion of its course, and the retention appendix which holds barium long after the bowel has evacuated its contents.

#### DISCUSSION

DR. W. A. BASTEDO, New York City: It seems to me that in regard to colon irrigations we should obtain as much information as possible from the accumulated experience of the men here. In using colon irrigations we employ the double tubes. In a number of instances when we have used the single tube there has been some reaction which we did not get when using the double tubes, for example, nausea or distress or pain. As a consequence a number of patients have expressed preference

for the double tubes. Occasionally one prefers the single tube, especially if the anus is sore. I have thought that in the use of the single tube, the average nurse's tendency is to let too much water run in, so that the colon becomes distended and the patient is made uncomfortable. It has seemed to me that in a number of these instances, the irrigation material has probably gone up into the small intestine, a result usually not desired. The average nurse is not good at irrigation work; probably with an expert either the single or the double tube method is satisfactory.

We have irrigated for some years and have paid much attention to the technic of the irrigations. We have tried to clear out the cecum; and so far as we could tell both subjectively and objectively, the water has reached the cecum. At times after the cecum is apparently cleaned out the water will suddenly become filled with putrefactive material as if it had come from the small intestine; and I believe that in a number of instances the small intestine will empty its contents into the cecum during an irrigation. An important point in giving irrigations is to have the bag low, only a foot and a half or so above the patient, so as to allow the irrigation to proceed very slowly and without much pressure.

There is one case that I wish to tell you about, because apparently the characteristic mucus of mucous colitis is formed in the small intestine. The patient was operated upon some years ago by Sir Arbuthnot Lane, the colon being removed and the ileum attached to the sigmoid. That patient today, though without a colon, passes the ropes, strings and plaques of mucus so typical of mucous colitis. This case gives support to Dr. Herter's opinion that the lower ileum might be the important site of trouble.

DR. CHARLES G. STOCKTON, Buffalo, N. Y.: It is a fact well known to all of us that at Plombières, in the French Vosges, they have carried out a system of treatment of the bowel by means of repeated small enemata. I have seen results in the relief of mucous colitis that to me seemed quite extraordinary, by these repeated small enemata, never above 500 cc., which were repeated from five to seven times in the course of the forenoon. These small repeated enemata constitute a familiar line of practice now, as they have for many years. On the Continent, they regard the cure at Plombières as a very definite thing. I think something is to be gained by repeated small enemata with the double tube that cannot be obtained in any other way.

DR. JACOB KAUFMANN, New York City: Dr. Stockton's remarks recall an experience I had, a few years ago. Drs. Meltzer, Burton Opitz and myself were invited to see the exhibition by a Hindu of the methods that the natives employed in the district which he came from, in the Himalayas. He introduced a piece of hollow bamboo into his rectum, and said that was the way they do in India. They go into the river with the piece of bamboo cane in the rectum, and suck the water in, and push it out again. This is a crude method; but he exhibited it sitting on a bowl full of water. He sucked all the water into the colon, kept it in a while, and then let it out again. That was their method of cleaning out the colon. He laid stress on it, claiming that with it they kept up their good health.

With regard to the method of doing colon irrigations, I would say that I am still using a method which I got from Naunyn over thirty years ago; and I think it is practical. I use a large sized rectal tube, and insert it only a short distance. There

is no need to insert a great part of the tube because, as was demonstrated by X-ray examinations the tube passes only a short distance up the rectum and when more is introduced it coils up. Attached to the rectal tube by a connecting glass piece I have a large sized rubber tube and attached to this a large glass funnel. With this apparatus you perform the colon irrigation just as you would a gastric lavage.

One more remark about mucous colitis, which Dr. Bastedo mentioned: I should like to call attention to an excellent book published lately by an English investigator, Robert McGarrison (*Studies in Deficiency Disease*, Oxford Medical Publications). Stationed in a part of the Himalayas where they had plenty of wild monkeys, he took a number of them, put them in cages, and let them feed on the food that they were accustomed to. An equal number he put on food that human beings are accustomed to take, particularly meat. All of the second set developed constipation and chronic colitis, with discharge of bloody mucus. That is what you get by indulging in diets, which civilization gave to us.

DR. JOHN BRYANT, Boston, Mass.: Dr Kaufman's remarks on the habits of the Hindoo recall Pliny's statement in A.D. 77, that the ancient Egyptians ascribed the discovery of enemata to observation of the habits of the sacred Ibis, Pliny recalling the fact that this bird "washes the inside of his body by introducing water with his beak into the channel through which our health demands that the residue of our food should leave."

With regard to treatment of the hyposthenic type of patient with large right colon and spastic left colon, I have found it useful to divide them into two classes on the basis of the presence or absence of ileal regurgitation, as proved by roentgen examination. I am aware that many competent observers consider ileal regurgitation as a matter of no importance. I can only say that my personal experience is to the contrary. On the basis of immediate prognosis there is perhaps little difference in regard to the satisfactory reaction of these two classes of patients to simplified diet. There is, however, a very marked difference in the prognosis as concerns later recovery, for under the same method of treatment, a patient without ileal regurgitation will regain health much more rapidly and completely than one in whom the presence of ileal regurgitation has been demonstrated.

It is possible to bring the majority of these hyposthenics back to a reasonable degree of health without resorting to the use of enemata at all. One of the most satisfactory methods available, is the use of simple non-putrefactive food, by which is meant in general the kind of diet that one would give a small child. The patient should be kept upon this simple food, with eggs, meat and fish practically eliminated for from six weeks to two months. At the end of this time, several distinct indications of progress should be in evidence; among these may be mentioned the fading out of the dirty yellowish color of the skin, an improvement in the local gastrointestinal condition, and a distinct decrease in the occurrence of manifold nervous disturbances, these being frequently of a very bizarre type, such as hot spots on the head which can be definitely outlined, and which should disappear completely in the course of treatment. Having decreased what may be called the surcharge of nervous irritability due to the ingestion of unsuitable food over a period of years, one is then left with the problem of controlling the remaining and fundamental hyperirritability of the central nervous system of the patient. In connection with diet, it may be mentioned that unless one takes the trouble to increase the tone of

the intestine before giving agar, the difficulties of the patient may be increased rather than decreased as a consequence of the increased bulk which the already overworked intestine must dispose of.

A second important factor in treatment is the question of developmental physical exercise. If one stops to consider it, it is self-evident that the viscera are really placed in a sling; the diaphragm makes one end of this sling, and the other is represented by the lower abdominal muscles. These are really compensatory breathing muscles since by their contraction they may force the diaphragm toward its highest position of full expiration. Examine the long thin asthenic fluoroscopically in the standing position. His diaphragm will be without tone, and at its lowest position of extreme inspiration. Such a patient must in effect exhale before he can inhale. In other words, he must by contraction of his lower abdominal muscles force his diaphragm upwards before it can descend in its normal inspiratory excursion. Since the tone of the diaphragm, as of most of the patient's other muscles is usually deficient, I always begin by giving such an asthenic patient exercises in the lying-down position. The tone of the diaphragm may be greatly improved in this way just as the tone of the main skeletal muscles may be greatly improved by systematic exercise. Repeated fluoroscopic examination also gives assurance that there is a corresponding improvement in the tone of the intestinal muscle itself. Although the patient with ileal regurgitation may always require to be upon a somewhat limited dietary, the other class of patients without ileal regurgitation may expect that when the tone of the intestinal and skeletal muscles is reasonably restored, they may be able to disregard diet, provided always that they keep themselves in fair physical condition by a sufficient amount of general exercise. One has only to listen to the commotion resulting from active alternate contractions of the diaphragm and lower abdominal muscles, to be convinced that this special method of exercise is a valuable factor in the stimulation of intestinal tone. It is also a method which is very gratifying to the patient because unless deaf he often has immediate audible proof that he is getting positive results.

DR. JEROME M. LYNCH, New York City: My experience does not coincide with that of Dr. Rehfuess. I have found that as long as you irrigate the colon, the patient feels all right; but that he goes back again as soon as you stop the irrigations. It is the same result as that accomplished by diet, exercise and so on. I think that irrigation of the colon is attended with great danger when done by anyone not qualified to do it. One man in New York is doing a land-office business in irrigating colons. People sometimes get an idea from their family physician that irrigation of the colon is a cure for all ills. I can readily understand that when a non-fusion of the cecum exists the cecum dilates every time it gets filled with gas, and is apt to kink; and I have operated on one patient for obstruction and found the cecum adherent to the liver by a plastic adhesion. In ordinary distention with some angulation to account for it, I think irrigation would accomplish the purpose. The people who come to me oftenest are those suffering from rectal constipation. People neglect themselves and after a while develop a tolerance; in many due to an enlarged sigmoid. When the sigmoid retains this material, a loss of rectal tone is apt to follow. Under such circumstances it is easy to understand why such people are constipated.

DR. MAX EINHORN, New York City: I think that very often a great many types of constipation are due to insufficient nutrition. The patients are afraid to eat, because they are constipated. If they do not eat, there is very little material in their intestinal tract; and the constipation is enhanced. By letting them eat, the food that comes in pushes the other out. No matter what food they take, this is the result. So simple feeding is of great importance.

A second point is that in spastic constipation, olive oil enemas are often of great use.

DR. FENTON B. TURCK, New York City: It was before this society in 1900 that I presented the results of my experimental and clinical researches on the "Treatment of the Abdominal Viscera through the Colon." Before this period, in the *Journal of the American Medical Association* (June 22, 1895; October 7, 1899) and *New York Medical Journal* (March 13-20, 1897) and in other publications, I had demonstrated by experimental research and clinical application the effects of "fractional colonic lavage." I also gave the "interpretation of results" of different irrigation procedures including "continuous irrigation" methods. The clinical reports of intermittent distention of the colon with water and with air, and the effect of heat and of cold in the colon with the "interpretation of results," were reported on a large number of cases (*Jour. Amer. Med. Assoc.*, May 5, 1900; *Med. Record*, October 7, 1905). It may be that the essayist had not sufficiently reviewed the literature for it appeared as if the speaker referred to this method and results as if it had not been known before. I must add that I believe that experimental work in animals gives us more fundamental facts or "points to be observed" on which we can base our clinical work. The clinical results are then corroborative and give a better "interpretation of results."

My experimental research and clinical application of the treatment through the colon, affecting the splanchnic circulation and the organs, is of great moment at this time. The effects produced on general metabolism are of wide significance. My results shown in the production of increased resistance to disease, of increased natural immunity, has been corroborated recently by a number of workers in France and in Germany. This renewed interest in this work has not only confirmed my earlier and more recent findings, but has awakened an interest in exact methods and technique which I have used for the past twenty-five years. It may be stated briefly as follows:

The use of heated water (approximately 300 cc. at a time) at high temperature (50 to 55°C.) injected into the colon. Removal of the water through the same tube and injection of additional heated water 50 to 55°C. This promptly affects the splanchnic circulation—intestines, stomach, liver and lungs. From the constantly recurring heat applications within the colon it results in producing the heat reaction in all the cells of the splanchnic area and causes greater metabolic and functional activity of these cells.

Repeated injections of the hot water after removal of the portion in the colon cooled by the body, maintains a maximum stimulation. This is repeated until the maximum heat stimulation or cellular reaction is obtained. When the height of this heat effect is produced, cold water is injected into the bowel which also acts as a stimulant to the cells and excites functional activity of the splanchnic organs. The previously heated body prevents any shock from the cold water injected. To further stimulation of this functional activity, air is introduced into the colon with

a bulb like a Politzer bag. Inflation alternating with deflation of the colon is practiced until peristalsis of the entire colon from cecum to anus is produced and the flow of the retained air and fluid out of the colon through the tube is established. This I termed "pneumatic gymnastics of the colon." This expulsion of the fluid and air by the induced peristalsis, removes the pressure of the air and fluid and results in a secondary rhythmical peristaltic action. The whole splanchnic circulation is simultaneously affected which in turn, improves the general condition. The improvement in the capillary circulation will be noted by the general improvement of the colon, of the peripheral surfaces, skin and mucous membrane. The metabolism of the body was found to be highly stimulated with increased natural immunity. These entire biological and physiological phenomena have been carefully worked out in my experiments on animals and compared with similar clinical observations made in patients.

DR. MARTIN E. REHFUSS, Philadelphia (closing): The only reason I brought this question before this Society was an obvious one. I cannot find out anywhere that there is a definite rule about this secretion.

Dr. Einhorn has made the statement that the fasting stomach is a dry stomach, whereas every text-book shows that a definite amount of titratable acid is found in the stomach. And there is no question—in scientific publications everywhere they seem to agree with the fact that there is some secretion in the fasting stomach, and I can say that in examinations of 30,000 people I have never got an absolutely dry stomach unless I did not get the tube in place. But the fact remains that there is secretion which can be shown without the tube. Now the question comes up whether the tube produces secretion. We started in with a wooden tube and a glass tip and a metal tip and we satisfied ourselves first that a tube passed that way or a simple catheter would show the same sort of phenomenon. Now there is a great difference in different people. In going over this we precipitated the same discussion in case of ulcer. I agree that in cases of ulceration the acidity does return after three to five years. I did not make that statement but said that cases gave that psychic secretion, after he had finished the cure we were unable to get the psychic secretion. I say that that is a valuable thing, one of the valuable things is a reduction of the psychic phenomena which is compatible with the ordinary acidity two or three years later.

Now the question of egg white comes up—that acts differently in different people. If you take hypersecretory people and normal people, you will find that egg white is followed by a large secretion in the former, and in the other type of people you will find that there is no secretion following egg white, and that might be explained by the statement that Dr. Crohn has made.

Now the question comes up as regards the regurgitation—I do not know anything about that except that there is apparently such a curve, and that is what we are here to find out, and some of the members of this Association are better able to find that out, but it does occur, and if you take Spencer's curve, you will find that there is an regurgitation which is active during digestion. I can say that there is also pathological regurgitation which might give rise to term tryptic regurgitation.

The question about digestive type—I spoke about that. Unfortunately I did not measure the length of the epigastrium, but my impression is that the fast stomach occurs in about 40 per cent and the slow stomach occurs in about 30 per cent of normal types being predisposed to a slow evacuation.

## SOME UNDERLYING CAUSES IN THE PATHOLOGY OF THE COLON

JEROME M. LYNCH

*New York, N. Y.*

Previous to the sixth week of fetal life, the small and large intestine are structurally similar; in fact, the small bowel a little more advanced than the large.

After this period, the line of demarcation becomes definite. A bud appears on the posterior limb of the U shaped tube, and from this bud comes the cecum and appendix. And in its development from there on is determined normalcy or the foundation of pathology.

The large intestine now assumes the advantage of position and develops much more rapidly than the small intestine. With this development there is a retrogression of the rudimentary digestive apparatus; the villi flatten out and disappear, eventually forming follicles or inverted villi. It is significant that an organ primarily endowed with potential digestive possibilities, should gradually degenerate into a more or less useless rudimentary organ. This might be considered the primary critical period in colonic development, filled as it is with possibilities for harm or good. The cecum and sigmoid in their development are as variable as the wind, and may assume any size within the space limit in which they are enclosed.

It is my opinion that an overdeveloped sigmoid is unquestionably one of the fundamental underlying causes of gastrointestinal digestion and colonic pathology; and that surgical correction of this deformity is followed by brilliant results.

The liver, having the advantage of position in early fetal life crowds most of the intestines outside of the abdominal cavity. After the liver loses its advantage and the abdominal cavity develops, there is a gradual recession of the intestines within this space. It is during this period of recession that failure of rotation may occur, leaving the large bowel entirely on the left side; so we may call this the second critical colonic period.

After rotation takes place, the bowel takes its position over the right kidney. Here, again, is another critical period, since adhesions may so form as to result in elbow deformity of the colon later on.

During late fetal stages, and about the time of birth, the ascending colon, which is then suspended in the mesentery comes in contact with the right abdominal wall and a fusion, or adhesion takes place. In cases where the cecum does not descend to the right, iliac region, it remains free and the colon, immediately above it, may then fuse in the region of the liver, or actually to the under surface of the gall-bladder.

It would appear that there is a stage where the parietal peritoneum tends to form adhesions with the visceral peritoneum and mesentery of the colon that comes in contact with it. The covering or peritoneal coat of the gall-bladder and liver seem to tend toward fusing with the mesentery of the colon in a similar way. The other mesenteries at this time do not come in contact with the posterior wall of the peritoneum.

Other mammals, it is reported, the dog, cat, etc., do not develop these posterior wall fusions of the ascending and descending colon; probably because their posture is such that the mesenteries do not rest on the post-abdominal wall. Some humans, similarly, never develop these fusions of the ascending colon to the posterior wall and the large intestine from cecum to descending colon is then free in the mesentery. In such cases the liver and gall-bladder sometimes adhere to the mesentery of the colon.

This free colon is simply as if the transverse meso-colon suspended the large intestine from the cecum across to the descending colon, which, if free at all, is not so loose as the right part of the colon.

These cases are not to be confused with the entirely different condition of the first part of the duodenum being attached to the gall-bladder or liver by an extra continuation caudally and to the right of the gastro-hepatic omentum which usually ends where it passes from the first part of the duodenum to the porta-hepatica and surrounds the hepatic artery, bile duct and portal vein.

Dean, Stockard, and Bryan have suggested that human beings may be divided into two classes, with intermediate types, classified as meso- and hyper-ontomorphs. Stockard simplifies these designations into high and low thyroid types. The studies of these gentlemen have assisted materially in classifying many problems connected with alimentary pathology that were heretofore obscure; and they have also helped us to deal more intelligently with these different

types. We must bear in mind, however, that there are individual idiosyncrasies and not everyone can be labelled according to a cut and dried classification.

It has always seemed to me that we must take into account the fact that though we start originally with two simple cells, these two cells by their process of evolution evolve a most complex human being. I have in mind particularly the possibility of structural weaknesses of various tissues that may eventually lead to pathologic changes. For instance, there is the variable quality of connective tissue, and as this tissue enters into almost all the structures of the body we realize the possibilities for harm.

I can hardly believe any of us feel that the explanations so far advanced as to the etiology of diverticulosis are satisfying. It is well established that wherever a blood vessel passes through muscle it is surrounded by a protecting ring of connective tissue; and as diverticuli usually occur where blood vessels pass through muscular tissue, would it not be logical to conclude that diverticulosis is individual and a result of defective quality of connective tissue?

We have learned, especially from the studies of Bean, that the high thyroid type has a short, small intestine, and a rather bulky and long, large intestine, the low thyroid being the opposite and this difference explains why one type is more subject to colonic diseases than the other since a long and loose, large bowel is more difficult to handle than the short, compact bowel of the low thyroid type.

If my memory is correct, the non-specific infections of the bowel occur most frequently in the high thyroid types. This would be logical, as a static bowel is more liable to infection than one not static.

You are all familiar with the almost insuperable difficulties one meets in investigating the origin of non-specific infections on account of the varied flora of the colon. I have, therefore, been deeply interested in 4 cases that I have seen in recent years, 3 of which suffered from a purulent infection of the colon. The cultures from these 3 cases showed a pure, colon growth; the pus had all the characteristics of colon infection. In the fourth case, the patient had suffered for years from recurring attacks of toxemia, fever malaise, with alternating attacks of constipation and diarrhea, the urine always giving a pure culture of colon bacilli. In culturing his feces we got the same result. Though this man is on a diet consisting largely of acidophilus milk there is no change in the flora. This man is a high thyroid type, and this is typical with our experience after an ileostoma showing the capacity of the bowel to accommodate itself to change the conditions.

However, this is not the particular point I wish to present. What I have in mind is the possibility of an organism that is constantly present in the colon—namely, the colon bacillus—being the underlying cause of colonic infections. The discontinuous or segmental character of some infections is rather puzzling. And this, again, brings up an interesting problem as to why some simple infections result in stricture, while others, far worse, escape. To study the underlying causes, one naturally thinks first of the fact that the nerve supply of the intestines is developed segmentally, and to the variability of muscular tone. We may have, for example, spasm of a muscle due to increased blood supply or irritability of the nerve endings. These facts may throw some light on the segmental character of infections. We have all witnessed through the fluoroscope segmental spasticity, and we have palpated segments of bowel that felt like a piece of rope. I have in mind a patient under the care of Drs. Bull, Lambert and Tuttle suffering from right iliac pain, constipation, auto-intoxication, sleeplessness and nervousness. These gentlemen palpated a cecal tumor and made a diagnosis of presumable malignant tumor of the cecum. Owing to mere chance on my part, the diagnosis was cleared up and the patient recovered. During the absence of the attending physicians he came under my care. In order to empty his bowels, I ordered warm, ichthyol irrigations. Relief was almost instantaneous, and gave me a clue to the diagnosis—spasticity of the cecum. He had one other attack when he resumed smoking, evidence of individual susceptibility to tobacco.

Stockard believes that high thyroid types are more subject to alimentary cancer than the low thyroid. Of course, this is subject to contemplation. Personally, I believe cancer is individual, and that Stockard's theory that it is due to poor growth balance is more satisfying than any theory so far advanced.

Ileo-cecal insufficiency so long in the limelight, is a potent cause of alimentary constipation, with all that it implies.

Two factors enter into the etiology of ileo-cecal insufficiency, namely, sympathetic and mechanical. It is well known that splanchnic stimulation causes contraction of the ileo-cecal sphincter and relaxation of the bowel above it. Adrenalin, injected hypodermatically, will cause contraction of the ileo-cecal sphincter.

While not within the scope of this paper, the importance of sphincteric irregularities in colon pathology should be noted.

In this paper I will confine myself to the mechanical factors entering into ileo-cecal insufficiency. It is axiomatic that any interference with the contraction of a muscle causes it to lose tone. I have frequently observed this. Now the only cases where one might reasonably expect to remedy ileo-cecal insufficiency would be those instances where some mechanical factor prevented the proper contraction of the muscle.

Within the last year, I have noticed while operating for right side pathology, enormous accumulations of hard fecal matter in the lower 18 inches of the ileum, due to mechanical ileo-cecal insufficiency.

While primary tuberculosis of the bowel is comparatively rare, still we must take into account its occasional occurrence, especially in children, its sudden onset, closely simulating acute appendicitis, the fact that it has been mistaken for sarcoma, on account of the tumor formation in the right mesenteric leaf, due to glandular agglutination; and above all to remember if diagnosis is made at the time of operation, it is surgically curable. Case X illustrates this point.

The ceco-appendical junction seems to be, frequently, the starting point; from there it spreads to the colon and ileum. I have an idea that ileo-cecal insufficiency and iliac-stasis may be responsible for tuberculous infection in this region.

The nerve supply to the gastrointestinal canal is developed segmentally. Each zone or segment, as has been shown by Keith, has its own pacemaker, and its own rhythmical contractions. Disturbances in any segment are reflected in the others and will interfere with the normal propulsion of food along the alimentary canal. These interferences may be congenital deformities, or acquired. The congenital deformities may not result in pathological changes until adult age is reached, or they may be precipitated by improper dietetic habits.

Some of the most common congenital defects are ileo-cecal insufficiency; non-fusion of the cecum; elbow deformity of the ascending colon; adhesions of the gall-bladder to the transverse colon or cecum and over-development of the sigmoid.

The acquired deformities are those due to chronic appendix, pericolicitis, colo-cystitis, peri-sigmoiditis, and so on.

## THE EARLY DIAGNOSIS OF CARCINOMA OF THE UPPER COLON

MILTON M. PORTIS AND SIDNEY A. PORTIS

*Chicago, Illinois*

Carcinoma of the colon is not as rare a lesion as is commonly supposed. Unfortunately many of the cases are not recognized until it is too late for surgical relief.

This is not surprising for the tumors are of slow growth and metastases occur later than in carcinoma elsewhere in the body. Usually there are no symptoms in the early stages and if they do occur, they are vague and not localized. Hence they are frequently overlooked until some acute serious complication is added to the original lesion.

*Location.* These tumors are commonest in the sigmoid, next in the cecum and then at the hepatic and splenic flexure.

*Varieties.* Adeno-carcinoma is commonest, but other types like scirrhus and colloid are found. The carcinoma is frequently annular in its distribution but it may limit itself to one side of the bowel.

Pain is a very important symptom. Usually it is not complained of in the early stage, but later, owing to the inflammatory reaction in the colon occasioned by the tumor, pain of a dull nature is felt. This may be localized or diffuse. At times the pain is cramplike and severe, often with tenesmus. This is especially so when obstructive manifestations appear.

The patient complains of anorexia, nausea, loss of weight and strength and looks anemic. If these symptoms occur in a person past forty, the possibility of a latent carcinoma of the colon should be considered.

Tenderness somewhere along the colon is often found, but in the early cases usually a mass cannot be felt. When a tumor mass can be felt, it has great diagnostic value, but unfortunately the case has as a rule, advanced too far for surgical cure. Obstructive symptoms develop in most cases later on, but this again is a warning usually in a hopeless stage.

In the later stages, constipation alternating with diarrhea is a common story. The stools often show mucus and blood, but a profuse

hemorrhage is rare. The feces show occult blood in the early cases. If on a blood free diet, occult blood can be demonstrated in the stool, it has definite value in the diagnosis. We examine the stools in all of our cases as part of the routine of a diagnosis and follow up those that show occult blood in the stool very carefully. The diagnosis of carcinoma of the colon can thus be suspected earlier than by any other means. In case 1 where the diagnosis was made very early, it was this finding that called our attention to the possibility of such a lesion. The deformity seen by the X-ray in the cecum may well have been due to a scar from an appendectomy eight years before, but the repeated finding of occult blood was the deciding factor that led to the operation that saved the life of the patient. In case 2 occult blood was constantly present early and the X-ray did not reveal a lesion when the patient was examined by two excellent roentgenologists. We urged an exploratory operation but because of the negative X-ray results, the patient believed himself to be neurasthenic and went to Atlantic City for several months for treatment by a nerve specialist, and finally when acute obstruction occurred, operation revealed an inoperable carcinoma of the sigmoid.

The X-ray evidence is given best by Carman, who states that a filling defect is the most important sign. Next in importance is the presence of obstruction. An opaque enema observed by the fluoroscope is usually of greater value than plates. The examination is made by following the opaque meal through the colon and then giving an opaque enema.

The filling defect may be unilateral, or may appear as a concentric narrowing. Carman calls attention to the important fact that the filling defect of carcinoma may be imitated by a variety of conditions, such as gas, insufficient quantity of enema, fecal matter, spasm, extrinsic tumors, pressure of bony parts, adhesions and diverticulitis, tuberculosis and other lesions of the intestine.

In the differential diagnosis all other lesions of the colon and of most of the abdominal viscera must be included. According to the location of the tumor, the adjacent viscera must be considered in the diagnosis. Tuberculosis and actinomycosis and diverticulitis all must be excluded. A chronic appendiceal abscess may resemble a cecal carcinoma and must be kept in mind. Tumors of the gall-bladder, stomach, liver and kidney often resemble tumors of the colon but they can be ruled out by the laboratory and X-ray findings. An enlarged spleen or a displaced spleen or a wandering kidney also give confusing pictures and must be ruled out.

*Case 1.* Man, aged sixty-one, consulted us complaining of epigastric distress, loss of strength and anorexia. At that time he stated that he had not had his usual appetite for several months, but did not have any pain, nausea or vomiting. He would awaken in the early morning with an indefinite distress that he could not locate nor define. The bowels had a tendency to constipation; he had not passed any dark stools to his knowledge.

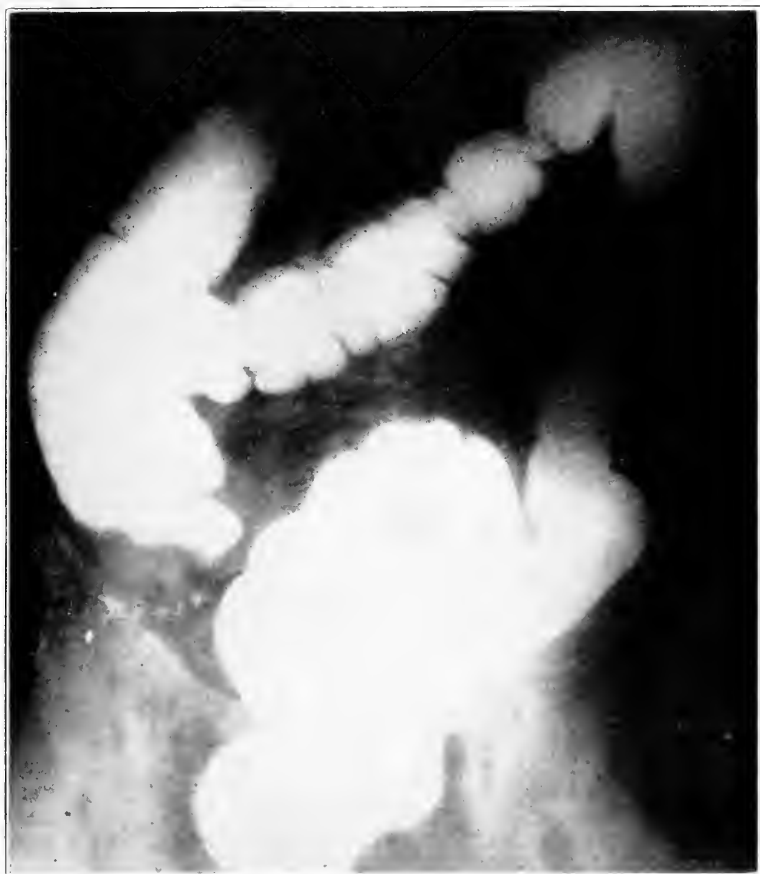


FIG. 1. SHOWING FILLING DEFECT OF CECUM

He stated that he had an attack of influenza during the epidemic thirty years ago, and that his appendix was removed eight years ago.

The patient when first seen showed a slight cyanosis, especially of the lips, but seemed well nourished. There were no abnormalities in the nervous system; the eyes were normal in all reactions. There was a slight emphysema of the lungs.

but otherwise the lungs were normal. The heart showed some enlargement to the left and right, the apex being 1 inch to the left of the nipple line. There was a systolic murmur heard all over the heart area and transmitted into the vessels of the neck. There was a definite arrhythmia present.

The blood pressure was 125 systolic and 73 diastolic.

The liver extended 4 inches below the costal margin; it was smooth and firm and not tender. There was tenderness in the epigastrium, but no mass could be felt. The spleen could not be palpated. There was also present an inguinal hernia of small size on each side. The prostate was slightly enlarged but not tender.

The urine showed a trace of albumin with a few hyaline casts, but was otherwise normal. *The stool showed definite evidence of occult blood.* The stomach test on repeated examination, including the fractional method, did not show any free hydrochloric acid at any time. There was no blood in the stomach contents.

The blood gave 3,984,000 red cells, 9500 white cells and a hemoglobin of 62 per cent. The blood Wassermann test was negative.

The X-ray examination by Dr. E. L. Jenkinson at St. Luke's Hospital gave normal lungs; moderate enlargement of the heart to the left and right; a slight diffuse dilatation of the aorta; early emptying of the stomach, but there was no defect and the duodenal bulb was normal.

The colon enema gave the following results: The rectum and sigmoid filled rapidly and showed no defect, the transverse colon was spastic, especially near the splenic flexure; the hepatic flexure was normal, the cecum showed a filling defect involving the outer and middle part of the tip (fig. 1).

Because of the filling defect at the cecum and the blood in the stool it was thought that the patient had a tumor of the cecum. The appendix had been removed eight years before and it was necessary to exclude deformity due to scar following the operation. In spite of the absence of the usual obstructive symptoms of tumor of the cecum, it was believed that the persistent presence of occult blood in the stool and the X-ray deformity of the cecum pointed to tumor, and operation was advised. Dr. L. L. McArthur resected the terminal ileum and nearly all of the ascending colon and then did a lateral anastomosis. On the inner surface of the cecum at the site of the old appendectomy scar a small cauliflower growth was found. Microscopic sections showed the typical picture of carcinoma.

*Case 2.* Woman, aged fifty-four, referred by Dr. Neff, complained of pain beneath the right hypochondrium for some six weeks. She thought she had injured her side doing housework. The pain was sharp and radiated to the shoulder and was aggravated by deep breathing. She never had been jaundiced. She belched a great deal but had no nausea. She complained of vertigo and headache. She was weak and had lost weight.

Physical examination showed a normal chest and lungs. The right upper quadrant was very tender and an indefinite mass could be felt. There was tenderness over McBurney's area. The urine was normal. *Occult blood was present in the stools.* The stomach test showed a slight hyperchlorhydria. There was a moderate secondary anemia; blood Wassermann negative.

The X-ray examination gave normal lungs and heart; stomach and duodenal bulb did not show evidence of any lesions. There was marked tenderness over the region of the gall bladder.

The opaque enema showed a filling defect at the cecum.

The patient was referred back to Dr. J. M. Neff for operation for gall-bladder disease and carcinoma of the colon, and he found a distended gall-bladder with calculi, which were removed, and an infiltrative hard irregular lesion at the tip of the cecum extending two-thirds around its circumference. This portion of the colon was resected and microscopic examination revealed a typical carcinoma.

*Case 3.* Man, aged fifty-seven came under observation November 10, 1919, complaining of pain, associated with belching and followed by nausea and vomiting, which brought temporary relief. The pain was felt in the epigastrium and came on two or three hours after eating; it was aggravated by taking sour substances and relieved by alkalies. During the last year he had had five such attacks, and had been given hypodermic injections to relieve the pain. The vomitus was clear and did not show any blood. He had never been jaundiced. The stools had always been normal in color. Although the stomach did not give severe trouble between attacks, yet he said he belched after every meal and at times felt considerable distress or discomfort in the region of the stomach. During the attacks he felt dizzy.

Examination showed some emaciation; patient was pale, reflexes intact; eyes reacted promptly to light and accommodation; nothing abnormal in the lungs or heart. The liver extended three fingers below the costal arch; not tender; smooth; of normal consistency. There was tenderness just below the liver on the right, but no definite mass could be felt. There was no tenderness in the region of the appendix. Small hemorrhoids present. There was no mass in the rectum and the prostate was normal.

The urine was normal. *The stool gave markedly positive tests for occult blood;* there was, however, no microscopic evidence of blood, and the stool was greenish-brown in color. The stomach test showed normal motility and the acid titrations were normal. There was no blood present. Red corpuscles 3,250,000; leucocytes 10,500; hemoglobin 58 per cent.

The X-ray examination by Dr. E. L. Jenkinson at St. Luke's Hospital showed a normal esophagus; the stomach normal in size and shape; no filling defects nor obstruction on the greater curvature; the waves normal; the pylorus regular; the duodenal bulb showed an irregularity at the apex; the stomach emptied in four and a half hours; the large bowel was flat throughout, the appendix flat, movable and not tender; the cecum and ascending colon freely movable.

The conclusion from the X-ray examination was that duodenal ulcer was present and this seemed more likely from finding blood in the stool on a meat-free diet. The patient was put on ulcer treatment for a week, but the bleeding continued and in spite of the negative X-ray evidence and because of the emaciation it was felt that we were probably dealing with a tumor of malignant nature and operation was advised. Dr. L. L. McArthur at operation, found a small tumor at the hepatic flexure of the colon. Because of the anemia and the poor condition of the patient, a two-stage operation was made; first, an anastomosis of the ileum to the transverse colon was made, and a few weeks later the entire ascending colon and one-third of the transverse colon was excised. The tumor was of the annular type and its surface was ulcerated; it was attached to the duodenal bulb; the microscopic sections showed typical carcinoma.

The reason that the tumor which was small in size and located at the hepatic flexure, could not be demonstrated by the X-ray, was probably that it was located in a fold of the colon and at the hepatic flexure, and was obscured by it. Possibly a repetition of the X-ray examination would have shown some filling defects in this region. The fact that the tumor was adherent to the duodenal bulb accounts for the deformity of the bulb.

*Case 4.* Man, aged fifty-two; under observation for eight years with cardio-renal symptoms.

In October, 1919, he began to have attacks of diarrhea and had some abdominal distress. The diarrhea yielded to simple measures but recurred two months later with more pain, which was referred to the perineum, and *occult blood was present on a meat-free diet.*

The X-ray showed multiple diverticulæ of the colon especially in the region of the sigmoid; there was no definite filling defect. While we were arranging for an exploratory laparotomy the patient decided that he was merely nervous and went to Atlantic City and placed himself for several months under the care of a nerve specialist, when acute intestinal obstruction developed and at operation Dr. Kanavel found an inoperable adeno-carcinoma of the sigmoid.

## DISCUSSION

DR. JOHN A. LICHTY, Pittsburgh, Pa.: In discussing this paper I should like to call attention to the fact that it seems to me the search for occult blood was stressed a little more than is justified. The malignant growths in the bowels do not always begin with ulceration, and there are conditions that might occur earlier than occult blood appears and should not be lost sight of. One of these is rather definite pain; pain which comes with defecation at times. The patient knows of it and usually describes it quite characteristically. It is different from the ordinary pain, it is very definite, and is associated with a definite point of tenderness. When this pain is present and there is a gradual increase in constipation, it is strong evidence pointing towards the suspicion of a carcinoma. It should lead to a definite decision as to operative procedure and should not be overlooked.

I should like to call attention to the fact that X-ray examination may be done under the best conditions, but unless the patient is prepared with a preliminary course of belladonna the X-rays are likely to be misleading. In fact it should be stated in reporting X-ray work on the gastro-intestinal tract as to whether the patient has been belladonnaized before the X-ray was taken.

In tumor of the intestinal tract, especially of the colon, the bismuth enema is sometimes of considerably more value than the ordinary bismuth meal. If with the definite symptoms of pain and tenderness at a definite point with an increase in constipation and a defect shown under careful X-ray examination, even without occult blood, the diagnosis should be made. Of course the presence of occult blood constantly is a very definite symptom as was pointed out by Dr. Portis.

DR. GEORGE B. EUSTERMAN, Rochester, Minn.: I want to speak of two points that Dr. Lichty stressed. During my service in the army, I met some doctors down South. One young man was at Anniston, where they did not have very good facilities. About a year after being in the service, they brought in an elderly man with pain in the left upper quadrant. We could not find any cause for the trouble.

My experience told me not to be in a hurry to send him home. We went over him several times with the fluoroscope. Finally, I told the doctor to leave him with me for study. I put him on a meat-free diet, and found occult blood. He had an adhesion that should have been operated on, any way. It was not spastic. The fact is that I had a hard time to induce the surgeon to operate; but I told him about these tests. He was operated on, and we found a flat adeno-carcinoma about the size of a dollar. The occult blood test sent him to the hospital, thus emphasizing its importance; but he would have gone any way, because of the pain.

DR. JEROME M. LYNCH, New York City: I think that this paper is very important, because carcinoma of the colon is very difficult to diagnose. Usually, when one feels a mass, it is an accumulation of fecal matter above the obstruction and not the growth that is felt. It is fortunate that this accumulation happens, otherwise early diagnosis would be difficult. I remember 2 cases in which I found a large tumor, but only a ring of carcinoma was found at operation. The patient is still alive, notwithstanding the fact that there was extensive glandular involvement. When the tumor is felt, the patient is usually beyond operation. One should make a careful examination in tumors around the ileocecal region, as they give very few symptoms except indigestion and perhaps pain. It has been my experience that they always have indigestion, and have seldom complained of pain. In one patient, a case of Dr. Robbins's of Richmond, he felt the tumor and thought it was in the sigmoid. Several of us who felt it thought the same, but when removed we found a small carcinoma involving the ileocecal valve which had migrated by intussusception as far as the sigmoid. Resection was performed, recovery followed.

DR. JACOB KAUFMAN, New York City: There is one sign not mentioned at all today in the discussion of the diagnosis of cancer of the colon, and that is, visible stiffening of the gut. There is no more reliable sign than that. When we see distinct stiffening of the colon we can be pretty sure that the stiffening is provoked by the effort of the gut to overcome a narrowing of its lumen. In cases where the history points to the possibility of cancer I have been able to diagnose the presence of a growth merely on the strength of this symptom, even in cases when the X-ray failed to show any evidence.

I have even had the experience that the surgeon would search for the colon cancer which I thus had diagnosed and failed to find it. I was not present at the operation in this case. Afterwards, the attacks of abdominal pain went on; and three months later, the abdomen was opened again by another surgeon, and a pretty big growth at the splenic flexure was found. Whenever the symptom of stiffening is present, it is more reliable than X-ray examinations and more reliable than the finding of occult blood, which may originate from many different sources, quite aside from the fact that in certain types of cancer, in scirrhus, for instance, no occult blood is found in the stool.

DR. MILTON M. PORTIS, Chicago (closing): Owing to the lateness of the hour, I shall not make any remarks on the points brought out in the discussion. I thank the gentlemen for discussing the paper. I want to emphasize one point. Someone spoke on the diagnosis of the condition, and one of the things stressed referred rather to the later picture of carcinoma. I had in mind the early diagnosis in which occult blood, rather than other symptoms, might have significance.

## TREATMENT OF SPASTIC CONSTIPATION

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It was Fleiner who first concentrated professional attention upon the distinction between atonic and spastic constipation. But while many authorities accept this division of chronic constipation into the two groups specified, others strongly dissent from this view on the ground that all the supposedly characteristic signs of spastic constipation are also occasionally observed in the atonic form. It is true that atonic constipation is by far the more common form, and in a considerable number of cases the two forms are associated; nevertheless the existence of a purely spastic type cannot be successfully disputed.

I shall confine this paper to a consideration of cases attributable to functional neurotic causes and the abuse of strong purgatives, disregarding for the present all the other causes of spasms of the colon, such as local inflammatory changes of the intestinal wall, adhesions, and pathological affections of remote organs.

In spastic constipation the retardation in the evacuation of the bowel is induced by a spasm (enterospasm) of a few isolated loops of the intestine. This spasm is brought about by an increased irritability of the vegetative nervous system, which may in turn be due to neuropathic conditions associated with diseases of the abdominal viscera or pelvic organs, or to either vagotonia or sympathicotonia from disturbance of the internal secretions.

The normal function of the intestine depends upon the innervation supplied by the two opposing systems of nerves, the vagus acting as the accelerator, while the splanchnics are inhibitory. In a general way we call the two systems, which are so wonderfully well balanced, the vegetative nervous system. Aside from individual variations which may take place under normal conditions, the several organic functions may undergo far-reaching changes through excess of tonus on the part of one or the other of these sets of nerves. Every increased stimulation of the vagus results in increased activity of the

muscles of the intestinal canal. Excessive stimulation, or vagotonia, induces spasm of the circular muscles of the intestine, contraction of the colon, and other phenomena.

Spasms of the colon are observed by means of palpation and roentgenographic examination. They occur most frequently at the transverse colon, hepatic and splenic flexures, sigmoid flexure, rectum, and anus. Spasm of the large intestine is important as the basis of spastic constipation, as distinguished from the atonic form. The characteristic symptoms of spastic constipation are delay in the fecal discharge, and an intestinal colic which usually precedes the defecation. In such cases there are various degrees of abdominal pain, with or without meteorism, which may affect the entire abdomen or only certain portions of the intestine. These pains are often continuous for hours, and finally terminate with the occurrence of defecation, which is often quite voluminous. It is frequently possible to palpate as a thick rope the tender contracting intestinal loops, particularly those of the descending colon and the sigmoid flexure. There is frequent desire for stool, with incomplete evacuation. Spasm may be associated with atony of the distal segments of the colon, and hypermotility or normal tonus of the proximal segments.

Spastic constipation is not to be understood as a mechanical obstruction to the passage of the feces through the intestinal canal; if it were so, hypertrophy and distention of the sections involved would result. High-grade spastic contraction is compatible with the forward movement of the intestinal contents, for it is not a permanent tonic condition. Not infrequently spastic and atonic conditions of the colon alternate or even occur at the same time, as is evidenced by the fact that the retention of feces in the ascending colon is accompanied by excessive tonus of other sections, such as spasms at the beginning of the transverse colon. When functional constipation varies—passing from one form into another, the movements of the colon presenting different conditions at different times, we group these several manifestations together as spastic constipation.

Spastic phenomena of the colon, with or without constipation, occur more frequently in women than in men, probably because of social conditions, the nervous status in the better situated classes, and the causal relation between intestinal function and diseases of the female genital organs. This is the case especially in pronounced neuropathic individuals who are subject to organic disturbances of the gastro-intestinal tract. In the neurasthenic, functional disturbances,

among them constipation, usually arise without spastic change of the intestine.

The most varied conditions of the stool coexist with spasms of the colon and of the rectum, as to form, consistency and frequency. In spasms of the colon with prolonged haustral segmentation, the fecal matter is in the shape of irregular balls, while in proctospasm it is cylindrical or ribbon-like.

By regularly palpating the abdomen and by roentgenologic observation, strongly contracted sections of the colon may be located, especially of the sigmoid flexure and the descending colon. It is easy to distinguish between contracted and filled coils of the intestine by examining the patient repeatedly and at various times of the day. The tonus of the anal sphincter shows the presence of proctospasm, a condition which accompanies spastic contractions of the colon. The characteristic physical sign of this disease is found upon exploration of the rectum, which, instead of being filled with feces, as is common in atonic constipation, fits closely around the examining finger (proctospasm), almost like the finger of a glove. The deciding factor in the diagnosis is the roentgen-ray examination.

In the treatment of spastic constipation, the first care is to provide physical and mental relaxation for these patients, who are usually in an overstrung nervous condition. We recognize that a hypertonic state of the musculature of the stomach and intestine is a constant result in vagotonia. The intestinal canal is unduly constricted. Complete rest in bed or a fresh-air treatment is often sufficient to induce normal defecation. The principal point, however, is the prescription of a suitable diet. I recall the case of a well known automobile manufacturer who had maltreated his intestine for years with coarse articles of diet and large doses of a variety of purgatives, with the result that he required large doses of cathartics every night. In spite of his objection and to his great astonishment I immediately stopped all the laxatives, prescribing as a first measure a mild diet, as will be detailed later on. In the morning of the second day there was a spontaneous normal defecation, an event which had not happened in several years.

The diet in the different varieties of constipation is, on general principles, to be formulated in such a manner that a mechanical and chemical stimulation will be produced.

An active diet rich in insoluble residue is particularly indicated in chronic atonic constipation. In such cases there need be no mis-

givings in advising large quantities of coarse food, so long as the stomach is in normal condition. In spastic constipation the indications point to a chemically acting dietetic regimen exclusively; all kinds of mechanically irritating foods are to be absolutely forbidden. In many cases it may be necessary to occasionally administer vegetables and boiled fruit in purée form, so that our only dependence will be on the mildly laxative chemical effects of appropriate foods.

In chronic atonic constipation a coarse diet, rich in residue, to render the feces more voluminous, is prescribed to incite peristalsis. By this large and bulky diet a mechanical effect is obtained which increases the tonus of the relaxed intestinal musculature. In spastic constipation, however, the treatment is directly opposite. A diet should be selected that will make but slight demands upon the intestine. Soothing, antispasmodic foods should be administered, while the use of water and foods acting mechanically to increase intestinal motility should be excluded. No laxatives should be given in the treatment of spastic constipation. Glycerin enemata and glycerin suppositories are strictly prohibited.

In spastic constipation raw vegetables and raw fruits containing coarse cellulose, cabbage of all kinds, mushrooms, coarse bread, Graham bread, fruit with small sharp kernels, such as gooseberries, currants and strawberries, should be avoided, as they are unquestionably likely to maintain or even increase the irritable condition of the intestinal mucosa, both mechanically and chemically. Strong spices, strong black coffee, alcohol, or carbonated cold beverages are not allowable. All beverages should be avoided which are known to have a constipating effect, such as strong tea, cocoa, claret, blackberry wine, etc. We must prescribe a soothing diet, endeavoring to render the feces pasty and soft and the intestinal mucous membrane pliant and slippery. Young vegetables, the cellulose of which is comparatively soft and tender, may be allowed, at first in the form of the finest purée and prepared with the yolk of an egg or butter. Fruit should always be stewed. Many dishes are easily made with soluble and easily digestible fats, such as cream, olive oil, almond milk, and butter. Soft fat cheese, cream cheese, and curds mixed with cream are best. Eggs are to be served soft-boiled or raw.

I prescribe a diet as follows:

*Breakfast:* Orange or grape fruit; porridge boiled in milk and strained, with sugar and plenty of cream and butter; weak or caffeine-free coffee, or buttermilk; two ounces of cream; toast, butter, honey, jam or fruit sauce.

*Luncheon:* A cup of mucoid soup; sardines in oil; tender vegetables with cream sauce; light egg dishes with vegetable purée and mashed potatoes; jam stewed fruit (strained) or honey; cream cheese; caffein-free coffee with cream; toast, and butter; lemonade sweetened with 2 tablespoonfuls of levulose.

*Dinner:* A dish of mucoid soup prepared with butter; 3 ounces of fowl, veal, pigeon, sweetbread, brain, tongue, tender fillet of pork or tender roast beef, or fish boiled with a free amount of butter sauce; potato purée boiled in milk and mixed with butter; macaroni prepared in butter; plenty of purée of spinach, yellow turnips, young green peas, cauliflower, or artichokes, with butter, cream or the yolk of an egg; purée of stewed fruit, cream candies, or light farinaceous dishes with fruit or cream sauce.

Mucoid soups are made from oatmeal, rice, wheat starch, potato starch, and corn starch. The grains are boiled four to six hours with water, passed through a fine sieve and again brought to the boiling point, butter or cream added, and suitably seasoned ready for use.

An immense variety of fruits must be classed among the laxatives, because of their chemical constitution. These are of particular value in cases of spastic constipation. They stimulate peristalsis partly because of their fruit acids and partly because they contain sugar, which is likely to increase the fermentative processes in the intestine. Oranges, baked apples, grapes and watermelon are valuable. Fruit and its active ingredients may be freely used as jams, jellies, fruit juices, grape juice, cider, etc. Other acids and acid foods are recommended, as citric acid, lactic acid, buttermilk, sour milk, whey, kefir (two days old), sour cream, yoghurt, vinegar, and kraut. The cases of chronic constipation reported cured by the administration of the bulgarian or acidophilus bacillus will on investigation prove to be cases of spastic constipation.

The most useful kinds of the various sugars are the easily broken-up milk-sugar and levulose. Honey induces fermentation and acts well. Easily melted fats, as butter, oil, and cream, not only have a mechanical effect, as has been mentioned, but also act chemically, stimulating peristalsis by means of the great amount of fatty acids they develop. Pure white bread is not irritating and is best. No specific directions, as a rule, need be given concerning the consumption of meat in cases of spastic constipation. Meat may be taken freely, though some authors recommend a restriction or even a prohibition of it, on the ground that it induces nervous irritability.

The dietetic therapy may often be advantageously combined with certain medicaments. Large amounts of fat are capable of acting in a beneficial manner, rendering the feces soft and smooth. A. L.

Benedict was the first to draw our attention to a similar effect from liquid petrolatum taken internally. These substances may therefore be employed advantageously to increase the effect of the diet. Liquid petrolatum is indicated when we desire to lubricate the entire intestinal tract and facilitate the passage of its contents. The feces are softened and under the microscope are found to contain minute globules of the oil. Too heavy an oil should not be used, for this fails to permeate the fecal material, a desideratum as important as lubrication of the intestinal wall. The oil is not absorbed from the alimentary tract, and even in large doses has no poisonous effect. It is useful not only as a lubricant but also for healing superficial lesions of the intestinal mucous membrane. It may be given in tablespoonful doses three or more times daily.

In spastic constipation atropin or belladonna is our sovereign medicinal remedy. Atropin paralyzes the peripheral ends of the autonomic nerves and relaxes the spastic intestine. The extract of belladonna can be given three times daily in 0.008 gram ( $\frac{1}{8}$  grain) doses. In some cases atropin must be given in full doses. It may also be administered in the form of the less poisonous preparation eumydrin, the methynitrate of atropin, in 0.001 gram ( $\frac{1}{60}$  grain) doses two or three times daily.

More recently papaverin, which is an opium alkaloid of the isochinolin group, has been recommended. According to Pal's investigations it is said to have an elective paralyzing effect upon the smooth intestinal musculature and therefore has therapeutically valuable antispasmodic properties. This preparation has been superseded by benzyl benzoate. My own experience with benzyl benzoate in doses of 1 to 2 cc. (15 to 30 minims) three or more times daily justifies its use.

If the therapeutic treatment, as outlined above, should not have quite the desired effect, recourse may be had to oil enemas, as introduced by Kussmaul and Fleiner. Fleiner's oil enemata are extensively used in the treatment of chronic spastic constipation. Fleiner recommends for this class of cases one injection daily of 250 to 500 cc. ( $\frac{1}{2}$  to 1 pint) of the purest olive oil. The oil is to be retained in the bowel for a considerable time; it is best to retain it over night if possible. Should discomfort during the night (meteorism, pressure) result, as may occasionally happen, the time of administration should be changed, the enema being given at six or seven o'clock in the morning while the patient is in bed; the oil is then to be retained for three

or four hours, thus producing the same laxative effect as if it had been administered at night.

These injections should be continued for several months, at first daily, later every other day, and subsequently twice a week. The results are so good that in many cases of spastic constipation actual recovery is brought about without any other treatment. The oil, by partially breaking up into fatty acids, stimulates peristalsis; and it has a soothing effect on the tense muscular tissue. Besides it lubricates the gut, softens the fecal agglomerations, and forms a protective layer upon any inflamed portion of the mucous membrane.

As a rule no discomfort is caused by the "oil cure," and the patients are at the time hardly aware of the fact that the oil is being introduced. It has not been ascertained definitely whether the oil passes beyond the ileocecal valve in all cases, but some patients experience the taste of oil after receiving a number of enemata. The only inconvenience caused by the oil enemata is the impossibility of avoiding the soiling of the bed and the bedclothes. The patient must remain in bed for at least an hour after the injection, without indulging in much conversation, coughing, or laughter. Should there be no spontaneous action of the bowels in the morning, a small lukewarm sodium-chlorid water enema should be given.

Massage is contraindicated in spastic constipation, and all energetic manipulation aggravates the condition. Nor is the use of mechanical vibratory massage of any value in this form of constipation. All abrupt changes of temperature and all forms of mechanical irritation are to be guarded against. Warm applications made to the abdomen frequently overcome the spasms. A Priessnitz abdominal bandage is applied over night, and a hot water bottle or electric pad placed upon the abdomen several times during the day.

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#### DISCUSSION

DR. BURRILL B. CROHN, New York City: I would mention the fact that I have never seen a result from the use of benzyl benzoate in spastic constipation. I am also very much disappointed in the use of atropin in chronic constipation. In an occasional case brilliant results encourage its use. I think that raw fruits are more efficacious than stewed fruits. For instance, apples and pears are more useful in breaking spastic constipation than any other fruit except grapes. They should be

eaten, skin, pits, core and all. If you eat only the core, and discard the pulp, you get the result required; but if you eat the pulp only, and discard the core, you get no results.

DR. LOUIS M. GOMPERTZ, New Haven, Conn.: Dr. Vorhaus and I have been doing some work on chronic constipation and *B. acidophilus*. While it is true that the acidophilus milk will overcome constipation, it is my belief that the large amount of milk sugar used is an important factor in obtaining this result. We do not use the milk but rely upon cultures of *B. acidophilus*. We recently reported 100 cases of chronic constipation treated by this method and positive results were obtained in 25 per cent.

However, the intestinal toxemia which coexisted was markedly relieved. I believe that *B. acidophilus* will play an important part in the treatment of chronic constipation.

DR. CHARLES D. AARON (closing): In the treatment with atropin or belladonna, I believe we should push the administration of the drug until we get its physiological effect. The drug has been given to many patients in too small a dose. In many cases, we cannot give these drugs, and benzyl benzoate acts well. I agree with the last speaker that *Bacillus acidophilus* will in the future be of great benefit in the treatment of cases of chronic spastic constipation.

## CARCINOMA OF THE PANCREAS: A CLINICAL STUDY OF 138 CASES

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I have observed repeatedly that disease of the pancreas is the most difficult of upper abdominal lesions to recognize. This is particularly true when pancreatitis or carcinoma of the pancreas is not associated with jaundice or with a well defined tumor, or both. Yet the pancreas is the most important organ of the accessory digestive tract, and in view of the fact that disease or disturbed function of this gland is invariably attended by gastrointestinal disorders, a consideration of the subject is always of interest to the gastro-enterologist. The deep-seated, comparatively inaccessible situation of the organ, with the fact that there may be little or no gross evidence of impaired function, even in later stages of disease, are the fundamental causes for difficulty in reaching a timely and proper diagnosis. Because of this, carcinoma of the pancreas is usually recognized by its effect on adjoining structures, a fact which is apparent when we consider the anatomic relationship of the pancreas to the surrounding structures.

The only structure that intervenes between pancreatic tissue and the surrounding viscera is peritoneum, since the pancreas has no true capsule. The head of the gland is molded on the side of the duodenum; the anterior surface is in contact above and on the right with the beginning of the transverse colon, without even the interposition of peritoneum as a rule. The anterior surface of the body forms a considerable portion of the bed of the stomach and is in contact with the pylorus. The left end of the body is in contact with the splenic flexure of the colon. The tail is in contact with the spleen and is in close relation to the left suprarenal gland and the left kidney. Such intimate relationship of the gland to its neighboring structures argues for a close developmental connection and one can readily see how gross and invasive lesions of this organ may involve the terminal end of the common bile duct, and ampulla of Vater, the posterior wall and pyloric end of the stomach, the duodenum, the hepatic flexure of the colon and the large blood vessels, including the portal vein. In the discussion

of the symptoms of carcinoma of the pancreas textbooks and even current medical periodicals more or less agree that the onset is usually gradual, the initial symptoms beginning with vague, or mild, digestive disturbances, followed by a deepening jaundice, loss of strength and weight, distention of the gall-bladder, and variable types of pain in the upper abdomen in persons of middle age. In our experience such a classical picture obtains in only about half of the patients with proved malignant lesions.

#### DIFFERENTIAL DIAGNOSIS

In a consideration of the differential diagnosis it will be convenient briefly to consider the lesions in the upper abdomen which are associated sooner or later with jaundice, and those lesions in which jaundice does not play a part, as our material naturally falls into these two clinical groups.

Common duct stone, largely in view of its frequency, must be given first consideration. The history of acute colic, usually repeated, accompanied by rigor and pyrexia, probably shock during impaction of the stone, often sudden and severe colicky pain felt deep in the right side, above and to the right of the umbilicus, radiating to the back and to the right shoulder, followed sooner or later by jaundice which may or may not persist, without marked loss of weight and strength, are characteristic features. Patients with such symptoms are invariably young and the jaundice rarely becomes a deep olive green, or black jaundice as it was called by earlier medical writers. It must be remembered that in a small group of these cases pain may be absent and the complaint characterized only by varying degrees of jaundice associated with vague gastric disturbances usually consisting of attacks of nausea and gaseous distress.

#### CHRONIC PANCREATITIS

The type of chronic pancreatitis associated with jaundice and a distended gall-bladder will closely mimic cancer of the head of the pancreas. In these cases the patients are in an earlier decade and the symptoms are characterized by vague pains in the epigastrium, often associated with fever and a gradually appearing jaundice. The loss of weight and strength is not so persistent and striking as in carcinoma. At operation it may be impossible to distinguish between the two lesions. Surgical indications are invariably the same and the ultimate diagnosis is often made by the subsequent course of the

patient. There is another type of chronic pancreatitis not well defined, in which patients suffer from irregular chronic gastric disturbances and more or less moderately severe, or mild epigastric pain, and nutritional disturbances. In elderly patients there may be marked reduction in gastric acidity and enzymes. As a rule the complaint is not sufficient to justify surgical interference, but medical treatment is only palliative. There are other histologic types of chronic interstitial pancreatitis and its subdivisions; they will not be discussed in this paper.

In carcinoma of the gall-bladder, not an uncommon occurrence, differential diagnosis is more difficult. Here a palpable tumor in the region of the gall-bladder, jaundice, more or less persistent pain, and loss of weight and strength in advanced middle life are in common with the symptoms of carcinoma of the pancreas. Magoun and Renshaw have recently reviewed 84 cases in the Mayo Clinic in which 38 cholecystectomies and 46 explorations were performed. In the majority of these there was an antecedent history of gall stone colic, and the females were affected 4 times as often as males, which is almost an inverse ratio to the sex incidence of pancreatic cancer.

Carcinoma of the bile duct, ampulla of Vater, and duodenal mucosa around the papilla are relatively uncommon, fortunately, because a differential diagnosis would be extremely difficult. In carcinoma of the ducts distention of the gall-bladder depends on whether the growth is low (supra-duodenal), or at or above the juncture of the cystic, hepatic, and common ducts. In the latter case the gall-bladder would be less liable to be distended and the liver not enlarged. Carcinomatous metastasis from organs situated in the peripheral portal system (stomach, colon, rectosigmoid, and so forth) often give rise to jaundice, enlargement of the liver, gastrointestinal disturbance, and associated symptoms. The successful determination of the site of the primary growth is usually decisive in differential diagnosis.

#### BILIARY CIRRHOSIS

Biliary cirrhosis of the obstructive or acutely infective type is easily understood. It exists in connection with gall stones, particularly those in the common duct, and jaundice is an early and continuous feature. In many of these cases the patients are not cured by the removal of the gall stones and biliary drainage owing to definite degenerative tissue changes taking place in the parenchyma of the liver, but as W. J. Mayo has pointed out recently, there is a second

type of cirrhosis accompanying certain chronic biliary infections which is not so well understood. In these it appears that either primary hematogenous infection of the bile ducts takes place or that there is extension from the chronically infected gall-bladder to the ducts. In this type the liver is large and the walls of all the biliary ducts are extremely thick. One case was observed in which the lumen of the common duct was reduced at least one-half by the deposit of connective tissue in the wall of the duct. Every grade of biliary cirrhosis is to be found in this chronic type. It may be accompanied by an enlarged spleen, and not infrequently by chronic pancreatitis, and chronic jaundice is the rule.

About two years ago Mussey reported from the Clinic 90 cases of carcinoma of the pancreas which has come under our observation previous to 1918. The majority of these cases were proved by histologic examination. Practically all the patients exhibited malaise, lack of appetite, and loss of weight and energy common to carcinoma of the digestive tract. Pain was an outstanding feature and was the chief complaint of 44 per cent. The presenting symptoms next in frequency were gastric disorders 24 per cent, and jaundice 21 per cent. Pain occurred in all but 10 cases (88 per cent) and was usually described as deep, dull, and boring, of moderate severity, peculiarly nagging and continuous, apparently without cause, generally very difficult to relieve, and occurring usually in the left upper abdomen. In 32 per cent of the cases it also radiated to the back, a lower incidence than that associated with cases of gall-stones, in which posterior radiation is found in 55 per cent. In 47 per cent of the cases the constancy of the pain was permanent and is an important characteristic of pancreatic carcinoma. In the remaining 53 per cent the pain occurred intermittently only. Jaundice occurred in 41 per cent and was constant in all but 5 cases. This incidence is at variance with Opie's report of 82 of 113 patients having jaundice. Eighty per cent of the patients were males, the average age was fifty-six years, the average loss of weight was 26 pounds, and the average duration of illness only a few months. In 31 of the 37 patients with jaundice, in agreement with Courvoisier's law, some enlargement or distention of the gall-bladder was found at operation.

Since Mussey's report 48 other patients have been observed in the Clinic, in all of whom the diagnosis was definitely established by operation, necropsy, or histologic examination. Thirty-five (73 per cent) were males, and 13 (27 per cent) were females. The average

weight loss was over 29 pounds, the loss of strength was invariably great, and as in the first series, the average age was fifty-six years; the youngest patient was thirty-two and the oldest seventy-four. Twenty-nine (60.4 per cent) are known to be dead. The average duration of symptoms was four and eight-tenths months, which is strikingly short. It seems to imply that cancer in this organ makes gross inroads before disturbances are serious enough to influence the patient to consult a physician or surgeon. The family history for carcinoma was negligible; typhoid, severe dental sepsis or syphilis seem more important as possible etiologic factors. Gall stones were likewise negligible and an antecedent history suggesting pancreatitis was noted in a few cases only. These 48 patients fall into two distinct clinical groups. In group 1 were 26 patients (54 per cent) without associated jaundice; in group 2 were 22 patients (46 per cent) with characteristic severe jaundice, pruritis, clay colored stools, and so forth. Patients in group 1 are obviously of greater clinical interest than those in group 2. Carcinoma in the head of the pancreas was definitely noted in 11 patients (42 per cent). This was somewhat surprising in view of the absence of jaundice or distended gall-bladder. In the remainder the growths were chiefly in the body of the pancreas, sometimes including or involving the tail. A palpable mass corresponding to the location of the pancreas, invariably irregular, firm, and immobile, was recorded in 16 (61.5 per cent). The clinical records showed very little disturbance of bowel or urinary function. Gastric analysis and roentgen-ray examinations were made in more than 80 per cent of the cases. As a result, based largely on roentgen-ray findings, two diagnoses were made of cancer of the stomach; examination revealing subacidity, retention of both the motor and barium meals, and filling defects in the luminal outline. Operation showed that the tumor had implicated some portion of the stomach in each case. In 3 cases the roentgen-ray examination showed that the mass was definitely extrinsic to the stomach. In 1 case the growth obstructed the duodenum, and in one it obstructed the small bowel. Only 8 of the 26 cases (30 per cent) were given a primary or alternative diagnosis of pancreatic cancer. The same number were given a primary diagnosis of gastric cancer, and these were followed by such diagnoses as aneurysm, tumor of the hepatic flexure, chronic cholecystitis, duodenal ulcer with obstruction, and intestinal obstruction. In only 1 instance was a distended gall-bladder found at operation. This finding was not possible preoperatively owing to the marked

tenderness and resistance of the patient's epigastrium. The fullest possible study should be made of these cases in order that a higher percentage of accurate diagnoses may be reached. This point is well illustrated by a recent case in which an elderly patient presented himself with a brief history of vague upper abdominal disturbance, associated with marked loss of weight and strength in a relatively short time, and a complete benign achylia on repeated examination of the gastric contents. Roentgen rays of the stomach were negative. There was an absence of occult blood in the stool on controlled diet and repeated examination. There was no palpable mass, no unusual tenderness, and no evidence of scleral or cutaneous icterus. On further examination, however, the stools were found to be bulky, greasy, and grayish-white. Examination revealed a marked excess of fat and a markedly lowered sugar tolerance. Operation was not advised and the patient died soon after reaching home as the result of a severe cholemia and inanition. Necropsy revealed extensive carcinoma of the pancreas involving the head.

All of the 22 patients in group 2 had jaundice. The head of the pancreas was involved in all, as shown at operation or necropsy. The gall-bladder was found to be definitely distended and palpable in 50 per cent, and always associated with a definite enlargement of the liver. In the remainder there was enlargement of the liver, or a mass corresponding to the pancreas palpable in all except 3 cases. A definite tumor corresponding to the pancreas in location, consistency, and mobility was recorded in 4. In contrast to the first group the diagnosis was either primary carcinoma of the pancreas or obstructive jaundice of malignant origin in all of the cases. Operation or necropsy revealed distention of the gall-bladder, biliary ducts, and occasionally the duct of Wirsung, in 90 per cent. Congestion and enlargement of the liver was definitely recorded in 60 per cent. Metastasis usually extended to the regional lymph glands, the liver, and occasionally the spleen. In a few cases extensive metastasis to distant organs was noted as the result of blood vascular invasion. Free fluid, variable in amount, was recorded in 10 cases. Physical examination definitely revealed its presence before death, or preoperatively in 4; one was a case of probably pseudochylous ascites, the result of extensive metastasis to the mesentery of the small bowel and to the lumbar and aortic lymph nodes. Generally speaking the gastric disturbances were chiefly nausea, flatulency, and occasionally vomiting, usually bearing no definite relation to eating unless the stomach was implicated in the

growth. In 2 cases death was caused by perforation of the growth into the gastrointestinal tract. In 1 of the cases the patient was a woman of fifty-two, with a history of five months' trouble. The splenic flexure of the colon, the cecum, and the mesenteric vein at the hepatic flexure were perforated by a carcinoma of the head of the pancreas, complicated by severe hemorrhage. In the other case the patient, a man aged sixty-four, with a history of eight months of a watery type of diarrhea, loss of weight and strength, and ascites, but without jaundice or pain, had perforation of the duodenum resulting in general purulent peritonitis from carcinoma primary in the head of the pancreas. There was metastasis into the tail of the pancreas and abdominal lymph nodes. In another case rupture of a gastric varix was one of the terminal complications.

Pain was often insidious in onset and constant; it was also surprisingly sudden and severe in onset, at first sharp and intermittent, later dull and continuous. It is generally considered that the characteristic pancreatic pain is usually deep at the left arch, passing around or through to the left subscapular region, but as a matter of fact the pain is variable, often in the right upper quadrant, radiating through to the right side of the back if the growth is in the head of the pancreas, or it is supra-umbilical and to the right, or across the lower epigastrium, therefore corresponding more closely to the anatomic situation of the pancreas. When there is icterus it usually becomes progressively worse; it never entirely cleared in our series of cases although it often fluctuated in severity. The olive green or black jaundice invariably implies malignant origin; the progressive and rapid decline in weight and strength is striking. The patients are afebrile, although in a few instances inflammatory reaction or localized peritonitis and perforation provoked fever, rigidity of the abdominal wall, and leukocytosis, and thereby confused the issue.

Glycosuria was present in 5 cases or just a little over 10 per cent, and this was the average for the whole series. In 1 instance symptoms of diabetes antedated the malignant syndrome by almost two years. The glucose tolerance was markedly lowered in all of these patients and in a number of others who did not show glycosuria. A history of glycosuria, even transient, besides a lowered glucose tolerance test in suspected cases of pancreatic carcinoma has a certain diagnostic value. Glycosuria in the presence of pancreatic carcinoma means extensive disease of the organ. It is remarkable how the gland may carry on its internal secretory function in the presence of extensive disease and with only a few islets remaining.

## PANCREATIC FUNCTIONAL TESTS

More disparity exists between the well established physiologic importance of the pancreas and our power of estimating its functional capacity in clinical medicine than in any other organ in the body. In attempts to diagnose the more common diseases of the pancreas we have relied mainly on the evidence afforded by physical signs and symptoms. Considerable clinical experience when fortified by the findings at operation or necropsy has been our best guide. Undoubtedly we would have obtained additional help in the nonicteric group by the employment of all available laboratory tests besides the routinely employed gastric and fecal analysis and roentgen ray. The time-honored method of examining for excess fat and undigested muscle fibers, after a Schmidt-Strassburger test diet, in the stool is still employed. Fatty stools are occasionally recognized by the naked eye. Simple microscopic examination of the feces may often suffice; but the disadvantage lies in the fact that extensive inflammatory or malignant disease of the gland may exist without evidence in the feces. Moreover, quantitative determination of fat in dry or moist feces is a laborious procedure. Then the estimation of the ferments in the stool, especially trypsin and diastase, after the method of Gross and Wohlgemuth was adopted. After about 100 estimations in normal persons and in persons with proved pancreatic disease, we were convinced of the variable and misleading information furnished. All these and miscellaneous other tests have been recently appraised in an admirable essay by Garrod. Now we are in the third and most hopeful phase of the problem. The contributions of Grassmann, Bondi and Salamon, Wohlgemuth, Strauss, Einhorn, Lyon, Rehfuess and Hawk, Friedenwald and Sindler, Crohn, Whipple, Dowden and others are familiar to us all. But the findings have been too variable and the conclusions too divergent to make the procedure trustworthy.

The method devised by McClure and his collaborators, which seems more conformative to the basic principles of the physical chemistry of enzyme action is essential to obtaining more uniform results in the examination of duodenal contents. The method is somewhat too involved to be universally popular, but the tests can be effectively and accurately carried out in a well organized laboratory. The results of our investigations in this direction will be the basis of a future report.

## CONCLUSIONS

1. Carcinoma of the pancreas is a disease of middle life, the average age of patients being fifty-six years. The average duration of symptoms is about five months, and over 75 per cent of the patients are males.

2. Gall stones and heredity play a negligible etiologic rôle.

3. Obstructive jaundice and distended gall-bladder are present in less than half of the cases.

4. Rapid and progressive loss in weight and strength is a common symptom.

5. The nonicteric group constitutes the greatest problem in upper abdominal diagnosis. The clinical evidence in all cases is more circumstantial than direct.

6. The pain and gastric disturbances as a rule bear no definite relation to alimentation; the former is usually more marked at night, it is rarely influenced by eating, and is aggravated by the prone position.

7. Laboratory methods to determine the enzymatic activity of the duodenal contents have until recently not been reliable.

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### DISCUSSION

DR. MAX EINHORN, New York City: I should like to say a few words. I want to lay stress on one feature, and that is that the appearance of sugar in the urine can be made use of occasionally in the diagnosis. I have a patient of that sort now in the hospital. He came to me six weeks or a month ago, and had lost 20 pounds in weight within two months. He complained of pain in the stomach, but not especially severe. He felt weak and tired out. I found his liver swollen, and thought that I could feel something, which I ascribed to the gall-bladder—an indefinite mass under the liver. I examined the urine, and found that he had 2 per cent sugar at that time. He had never had severe colic. There was a gradual development, and that put me on the track of thinking that there might be some malignant disease present. I sent him to the hospital and had all kinds of tests made, and nothing was found—no blood in the stools. But sugar was present in the urine. I examined the bile and the pancreatic juices. I found the bile not exactly clear, but there were no definite signs such as we would usually find in cases of gall stones and severe lesions in the gall-bladder. The ferments, however, were very much reduced in strength. They were not absent. Instead of the trypsin being 3 mm. it was only 1—very much reduced. Based on the finding of sugar in the urine, I thought we might have to deal with malignant disease of the gall-bladder, and pancreas. I advised an exploratory operation. The operation was done less than two weeks ago. Nothing was found in the stomach; no stones in the gall-bladder; but we found a tumor of the pancreas, an extensive one. I omitted to say that a few days before the operation, the same gentleman developed jaundice; and that was another reason that I was more strengthened in the diagnosis of tumor of the pancreas. There was no pain. I had another case of the same kind, in which I made the diagnosis of tumor of the pancreas, based on the finding of sugar in the urine. That is an important point. There are many cases in which we do not find sugar present. If we do find it, we should look out.

DR. BURRILL B. CROHN, New York City: I should like to compliment Dr. Eusterman on his study of pancreatic carcinomata. It is a difficult subject, one in which I have been interested for some time. I approached it from a somewhat different point of view. I think it is necessary here to differentiate two kinds of growths: those that occur at the head of the pancreas, originating in the papilla of Vater or

the duct systems, and those arising in the parenchyma of the gland. In studying tumors of the head of the pancreas, I have found that it was necessary to differentiate those that occur as a flat epithelioma of the papilla or ampulla of Vater or the mouth of the duct of Wirsung, from those primary in the gland proper. The former type of carcinoma is really an epithelioma, which must be differentiated from the other tumor that begins, not in the duct, but in the body of the pancreas. This former is a cylindrical celled carcinoma, which, according to my experience, pursues a course like that of an epithelioma—an epithelioma of the gastrointestinal tract. Its duration is about nine months. Its metastases are local, by extension into the pancreas or the neighboring lymph nodes.

The latter type of tumor that I mention is a primary tumor. That type is exceedingly rare. Recently, in discussion, the point came up of the relative frequency of the two types. A search of the dead-house material from the largest German hospitals shows that tumors at the head of the pancreas arising in the ducts constitute 3 per cent of the total carcinoma statistics—the other type primary in the gland proper, about 0.1 per cent; so that the former is 30 times as common as the latter. The latter, while growing rapidly, does not involve the ducts till very late and metastasizes to the lungs, liver and other organs. I should like to ask Dr. Eusterman a question that I have put to a good many surgeons. I think early diagnosis is possible by means of the duodenal tube. In the absence of a functioning duct of Wirsung, do we ever see the accessory duct of Santorini capable of functioning for it?

I have seen some of these cases very early; I have tried to induce the surgeons to do an operation that has been done about 15 times in Europe, consisting of a preliminary cholecystenterostomy, then a gastroenterostomy, and then a removal of the head of the pancreas and the implantation of the duct of Wirsung in the duodenum. It has been done 3 times successfully. I have never been able to tempt a surgeon in this country to do this operation; it is the only operation that offers a hope of life.

DR. JAMES TAFT PILCHER, Brooklyn: I wish to refer to two points that Dr. Eusterman has not spoken of in his paper; although probably incorporated in the paper proper. I have invariably been impressed by a peculiar facial expression exhibited by those patients with this condition. It is difficult to adequately describe this peculiar facies. In the case of cancer of the pancreas it is much more intensified than in the cases of chronic pancreatitis; but after being seen a few times is readily recognized, and may prove of diagnostic assistance.

A differential point, which, if I recall correctly, Dr. Eusterman did mention, is that in the case of cancer the history is very short, and is fully developed at the end of six weeks to four months; while the course of chronic pancreatitis is relatively long. In either case, the patient may or may not be jaundiced; but is more likely to be in the instance of cancer of the pancreas.

In the Jewish Hospital, in Brooklyn, they have had a very remarkable series of cases of acute pancreatitis; and Dr. Wm. Linder has called attention to the frequency and the peculiarity of the pain that the patient complains of as being of the greatest diagnostic import. It is most frequently on the left side, and that there is in many instances a tender area at the angle of the tenth rib behind, somewhat similar to that spoken of as a diagnostic point in ulcer of the stomach. There are

many of these cases undoubtedly being met with, unrecognized unfortunately until too late to do anything.

The operation that Dr. Crohn has spoken about, I have attempted many times, experimentally only. Unfortunately, possibly, for the patient or myself, I have never had the nerve to tackle it on the human being yet. Possibly, as we grow older, it may be accomplished.

It is only by a study such as Dr. Eusterman has given us, that we shall ever get anywhere, and have definite data on which to base future investigations.

DR. GEO. B. EUSTERMAN, Rochester, Minn. (closing): I did not take time to go into the details contained in my paper relative to fat in the stools, the enzymatic activity of the feces, etc., as I did not wish to take up the time of the Society because of the late hour, but as Dr. Einhorn has brought up the matter of sugar I wish to say something about it. In 4 of these cases sugar was definitely present; this means that in over 90 per cent of people with advanced cancer of the pancreas there may be no sugar. Sir A. Garrod, in an admirable essay pointed out the diagnostic importance of glycosuria. However, even though this may be transient the glucose tolerance test in suspected cases may bring out an important fact. Most of these cases have a markedly reduced tolerance for glucose and in several instances patients having an absence of sugar in the urine showed a markedly reduced glucose tolerance. The presence of sugar argues for an advanced condition because nine-tenths of the pancreas may be involved or destroyed and yet the other tenth carry on the metabolic function.

The type of lesion that Dr. Crohn has mentioned and written about is extremely rare in our experience, in fact I was surprised at the infrequency of primary cancer of the ducts or of the ampulla in our large series in view of the reports in the literature. Differential diagnosis is difficult but on several occasions we have suspected this type of lesion by the presence of persistent occult blood in the feces.

Dr. Pilcher has brought up an important point. I am not clear in my own mind what chronic pancreatitis in many cases is and this doubt is shared by other observers. The pain was frequently in the upper right abdomen and back as in gall stone disease, especially where the head of the pancreas is involved. Hale White, of Guy's Hospital, London, has emphasized this point and also showed that the head of the pancreas is involved in over 75 per cent. The pain may be low right epigastric, or in the region of the navel in the vicinity of the tumor. The important point that Dr. Pilcher has brought out, however, is this: when there is a pain present which radiates to the back, especially around the left side, deeply situated, almost continuous, more inclined to be worse at night and in the prone position one should think first of cancer of the pancreas or posterior wall of the stomach.

## STUDIES OF GASTRIC SECRETION AND OCCULT BLOOD

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Sufficient time has elapsed since the introduction by Rehfuess (1, 2) in 1914, of the fractional method of gastric analysis to allow for the accumulation of data concerning the clinical significance of this procedure.

At this present moment, the profession is interested in the value of this method as compared to the single test meal analysis, the interpretation of the various curves, the ease of its application, and its clinical diagnostic importance.

### REVIEW OF THE LITERATURE

The review of the literature presents a universal and deserving commendation of the originators' work, but also a confusion of statements as to its interpretation and value.

Rehfuess, Bergeim and Hawk (3) presented the iso-secretory, hyper-secretory and hypo-secretory type of curves as a general grouping for normal individuals, also concluding that there is no typical curve. "They discuss the possibilities of a similar curve on succeeding days in the same individual under the same conditions, noting that there are many complex factors regulating gastric secretion such as: physical conditions, mental attitude, occupation, diet and environment."

They state that if the test will not give the exact figures of the preceding day, it will show identically the same type of curve, which is the important factor.

Forty-five per cent of normals present hyperacidity, 42 per cent of ulcer cases show the same figure: no acid figures occur in disease which cannot be duplicated in health. There is no greater incidence of high acid figures in ulcer or any gastric disease which does not occur normally. Any type of curve may be present in health or in disease.

Alteration in acidities according to these authors are due to psychic factors, the swallowing of saliva, the emptying time of the stomach,

the character of the test meal, the accumulation of inhibitory and sedative substances, the regurgitation of duodenal contents (Rehfuss and Hawk, 4) and partial neutralization by the mucosa. Pavlov (5), Boldyreff (6, 7), Carlson (8), Rehfuss, Spencer, Meyer and Hawk (9) found that duodenal regurgitation is a constant factor in normal gastric digestion.

Crohn and Reiss (10) agree with the statements of Rehfuss and his associates and in addition called attention to the fact that in the process of swallowing the tube, some of the retching, which invariably occurs and no matter how slight causes a regurgitation of duodenal contents into the stomach.

They also state that the curves are not new and conclusive diagnostic criteria. They emphasize the importance of the type of the curve rather than the actual acid values.

Best (11), Talbot (12), Lyon, Bartle and Ellison (13), Friedenwald (14), and Morgan (14), agree with Rehfuss and his collaborators. Bennett (15) states that there is but slight change from day to day in the curves.

#### SUMMARY

Fractional gastric analyses curves while not characteristically normal or definite for pathological conditions are more easily secured and of greater diagnostic value than the single withdrawal.

All the quoted authorities agree that the single withdrawal represents but one point in the ever changing phase of the gastric cycle.

From their conclusions we must infer that gastric chyme is a homogenous mixture at the time of any withdrawal; which specimen represents the acid concentration of the entire stomach contents.

Wheelon (16) and Gorham (17) state,

This hypothesis is not based upon true physiology, the acidity of one portion as obtained by the fractional method may differ widely from the acidity of different portions of the remaining contents. In the so called fractional or other methods of gastric analysis, when only a small sample is withdrawn, the small portion removed may or may not be representative of the gastric contents remaining in the stomach.

In their work, they quote Pavlov (5), Cannon (18), Grutzner (19), Prym (20), Sick (21), Taussig (22), Rehfuss (23), Home (24), Eberle (25), Ellenberger and Hofmeister (26), Ellenberger and Goldschmidt (27), Scheunert (28) and Know (29) to establish the fact that gastric contents is not as a rule a homogeneous mixture at every stage of digestion but the acid values of the contents differ according to its

location, whether the specimen secured was from the fundic or pyloric region of the stomach, or from the outer or central portion of the bolus.

Gorham (16) aspirated 10 cc. portions in rapid succession until the stomach was empty, the acidity of these samples determined by the Toepfer method varied according to the location of the tip, which constantly changes owing to the peristaltic movements of the stomach.

Wheelon (16) concludes that the acid concentration of gastric contents is not, in the majority of cases constant in all portions of the stomach at the end of one hour of digestion. In the withdrawal of gastric contents for the purpose of determining the acid concentration, the type of the meal, the position of the tube tip, and duodenal regurgitation are factors which militate against the acceptance of "fractional curves" as indicative of the secretory function of the stomach.

As pointed out by Gorham (17), these are physiological factors which in a great part may be held responsible for the various "secretory curves" which formerly were considered functional alterations in the power of the stomach to form acid."

Kopeloff (30) demonstrated that repeated analyses on the same individual within a short period of time with all factors equal yielded different curves, these curves varied in the same individual as much as the curves of different individuals. The variation of the acid values yielded the same results. His interpretation was that a single fractional determination was insufficient evidence upon which to base a conclusion.

Bennett (15), after the study of 100 normal cases and striking a mean normal curve states that there is no justification for the classification of normal cases under such headings as iso-secretory, hyper-secretory and hypo-secretory types.

Crohn and Reiss (10) state that no relationship could be traced between duodenal regurgitation and a fall of acidity, as the acid value was sustained even after this event had taken place. The presence of mucus did not interfere with the secretory values.

Hicks and Visser (31) state that it is difficult to conceive that duodenal regurgitation is the factor of greatest importance in the reduction of high acid values of the gastric contents, they interpreted the presence of bile in the gastric contents as evidence of regurgitation.

Talbot (12) in conclusion notes that many phases of the gastric cycle as demonstrated by the fractional method have yet to be explained.

L. Von Friedrick (32) demonstrated that in cases with subacidity, he could produce almost normal curves by thorough mastication of the test meal.

An editorial (33) in the *Journal of the American Medical Association* states that it is evident that the stomach must be emptied at a definite time after a standard meal, which will be recognized as a return in principle to the single withdrawal.

#### DISCUSSION

It is agreed that the factors mentioned in influencing acidity are accepted, except for the question of duodenal regurgitation in which the burden of proof rests upon those denying that it reduces the acidity.

The remaining factors occur at irregular times and are markedly influenced by the physical and mental condition of the individual tested.

We agree with Wheelon and Gorham in the contention that gastric contents are not homogeneous mixtures, but in the majority of analyses different acid values occur at different times, and in different portions of the stomach.

It is recognized that in the use of the large tube, the position of the tip is fixed, while that of the fractional tube seeks its level by gravity.

Our conclusions are based on the repeated study of fifteen normal medical students, on whom at forty-five minutes after the meal we performed 15 single withdrawals with a 32-inch stomach tube to which was attached a 480 cc. Davol aspiration bulb. Complete withdrawal was attempted. The 30 repeated fractional curves were secured with the Buckstein modification of the Einhorn tube. Aspirations were made at twenty-minute intervals. The corresponding high points were checked by 10 cc. withdrawal with the Buckstein tube. In all, 230 analyses were made on these cases.

In the study of these cases, we recognized the opportunity of testing for occult blood in a variety of conditions under different methods of withdrawal and have included them in this study.

The meal consisted of 90 grams of bread and 200 cc. of water taken in the fasting stomach.

Determination of the free hydrochloric acid was made according to Toepfers method using di-methyl-amidoazo-benzol as an indicator, for the total acidity phenolphthalein was used.

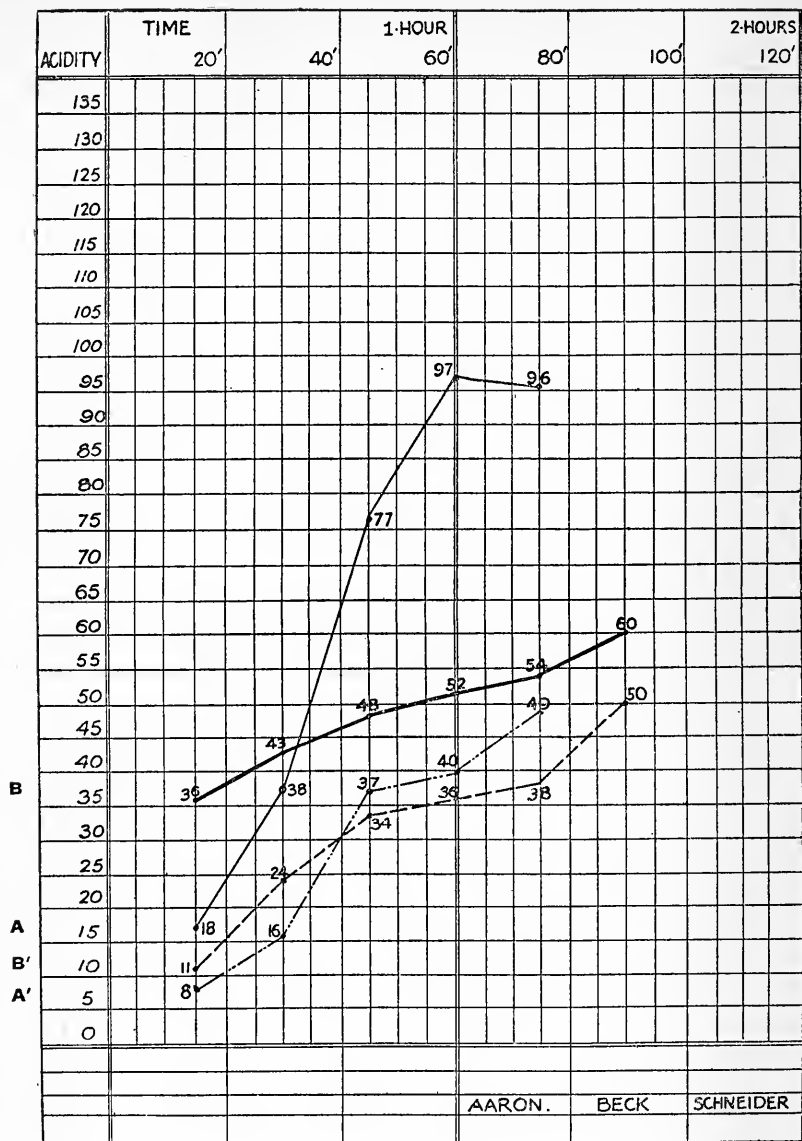


FIG. 1

$$\left. \begin{matrix} A \\ A' \end{matrix} \right\} \text{Curve of one test.}$$

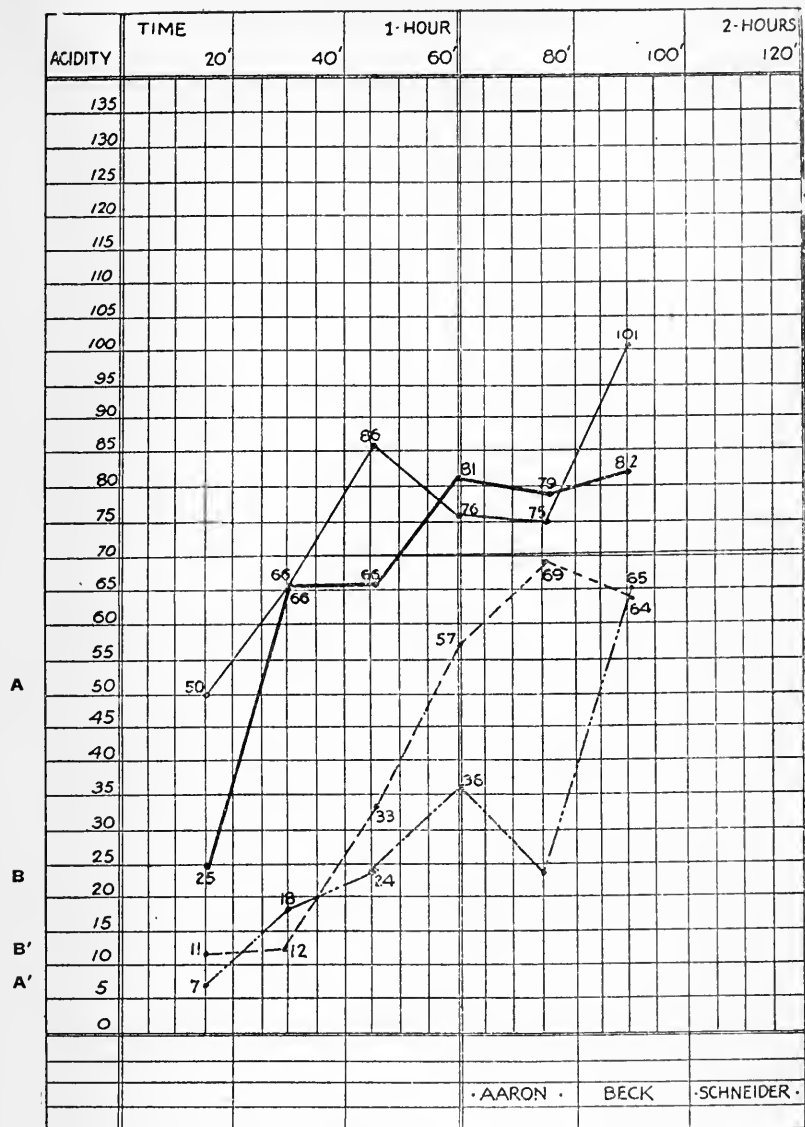


FIG. 2

Broken lines, Free HCl.  
Solid lines, Total acidity.

A }  
A' } Curve of one test.

B }  
B' } Curve of repeated test.

It was noted that the fractional tube in which a stylet was used passed more easily and caused less retching and discomfort.

Questioned as to which procedure caused the least mental and physical distress or discomfort, the ratio was 14:1 for the single withdrawal.

#### CONCLUSIONS

1. There exists a wide variation in acid values of gastric samples secured from the same individual at different times due to many irregularly occurring events.

2. Cases showing no acid or low values, may later show normal or high values by changing the location of the tip in the stomach and securing contents from a different location as is possible in case XIV.

3. The complete withdrawal by means of the fractional tube, a definite time after a standardized meal (preferably bread and water 45 to 60 minutes after ingestion) is easiest for the patient and gives valuable diagnostic aid.

4. Any secretory figure may be altered from day to day by physiological and extragastric causes to such an extent as to render one analysis be it fractional or single, of questionable value.

5. The absence or presence of free HCl is the most important secretory finding in the gastric contents, the actual figures vary to such an extent that only achylia or constant acidity below 15 is of diagnostic value.

6. Our repeated fractionals failed to show a characteristic curve.

7. 42 per cent of ulcers show hyperacidity  
45 per cent of normals show the same figure } Rehfuß.

17 per cent of cholecystitis cases show anacidity  
16 per cent of cholecystitis cases show hypoacidity  
12 per cent of cholecystitis cases show hyperacidity  
55 per cent of cholecystitis cases are normal } Hartman (34).

The distribution of these values renders the analysis of doubtful diagnostic aid, but of considerable therapeutic worth.

8. Pernicious anaemia cases show persistent achylia (Levine and Ladd, 35).

9. The use of a styleted fractional tube aids in its passage and reduces salivation and gagging.

10. The presence of occult blood in the gastric contents, obtained by a method which is carefully performed is of extreme diagnostic value.

| CONDITION                      | NUMBER | NEGATIVE | POSITIVE |
|--------------------------------|--------|----------|----------|
| Gall-stone . . . . .           | 4      | 4        |          |
| Cardiac . . . . .              | 2      | 2        |          |
| Pernicious anemia . . . . .    | 1      |          | 1        |
| Chronic appendicitis . . . . . | 32     | 32       |          |
| Post operative . . . . .       | 10     | 10       |          |
| Abscess of appendix . . . . .  | 1      | 1        |          |
| Cholecystitis . . . . .        | 16     | 14       | 2        |
| Cholelithiasis . . . . .       | 5      | 5        |          |
| Ptosis . . . . .               | 36     | 36       |          |
| Infected tonsils . . . . .     | 15     | 15       |          |
| Duodenal ulcer . . . . .       | 3      | 3        |          |
| Spastic constipation . . . . . | 1      | 1        |          |
| Colitis . . . . .              | 4      | 4        |          |
| Pylorospasm . . . . .          | 3      | 2        | 1        |
| Gastro-enteritis . . . . .     | 4      | 3        | 1        |
| Dilated cecum . . . . .        | 1      | 1        |          |
| Carcinoma of stomach . . . . . | 3      | 0        | 3        |
| Total . . . . .                | 141    | 133      | 8        |
| Normals . . . . .              | 90     | 90       | 0        |

All pathological specimens were secured with the large tube.

All normal specimens were secured with the fractional tube. Benzidine was used as indicator.

| NUMBER        | A  | B  | C  | D  | E  | C  | F   | G   | C  | H  | I  | C  | J   |
|---------------|----|----|----|----|----|----|-----|-----|----|----|----|----|-----|
| I             | 64 | 28 | 36 | 38 | 4  | 34 | 67  | 10  | 57 | 24 | 5  | 19 | 250 |
| II            | 74 | 83 | 9  | 44 | 39 | 5  | 110 | 100 | 10 | 91 | 66 | 25 | 0   |
| III           | 64 | 52 | 12 | 40 | 38 | 2  | 76  | 98  | 22 | 52 | 43 | 9  | 0   |
| IV            | 84 | 44 | 40 | 64 | 20 | 44 | 66  | x   | x  | 49 | x  | x  | 0   |
| V             | 55 | 85 | 30 | 40 | 45 | 5  | 97  | 76  | 21 | 61 | 58 | 3  | 0   |
| VI            | 58 | 52 | 6  | 45 | 17 | 28 | 68  | x   | x  | 28 | x  | x  | 0   |
| VII           | 83 | 74 | 9  | 63 | 48 | 15 | 98  | 85  | 13 | 78 | 55 | 23 | 300 |
| VIII          | 59 | 30 | 29 | 47 | 17 | 30 | 92  | 83  | 9  | 75 | 61 | 14 | 150 |
| IX            | 54 | 74 | 20 | 45 | 26 | 19 | 91  | 30  | 61 | 51 | 10 | 41 | 0   |
| X             | 68 | 55 | 13 | 48 | 34 | 14 | 100 | 61  | 39 | 64 | 33 | 31 | 190 |
| XI            | 70 | 70 | 0  | 50 | 48 | 2  | 90  | x   | x  | 68 | x  | x  | 0   |
| XII           | 54 | 40 | 14 | 38 | 18 | 20 | 70  | x   | x  | 48 | x  | x  | 0   |
| XIII          | 52 | 60 | 8  | 30 | 4  | 26 | 42  | 56  | 14 | 28 | 34 | 6  | 0   |
| XIV           | 68 | 0  | 68 | 58 | 0  | 58 | 80  | x   | x  | 65 | x  | x  | 150 |
| XV            | 60 | 90 | 30 | 50 | 35 | 15 | 90  | 75  | 15 | 73 | 57 | 16 | 0   |
| Average . . . | 64 | 56 | 21 | 46 | 26 | 21 | 82  | 67  | 26 | 57 | 42 | 18 | 208 |

A., 45 minute single withdrawal T.A.; B., Corresponding fractional point T. A.; C., Difference; D., 45 minute single withdrawal free HCl; E., Corresponding fractional free HCl; F., High fractional point T.A.; G., Corresponding single withdrawal T.A.; H., High fractional free HCl; I., Corresponding single test free HCl; J., Saliva in cc. T.A.—Total Acidity.

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## DISCUSSION

DR. W. A. BASTEDO, New York City: Did I understand Dr. Aaron to say that there was more occult blood with the fractional tube than with the single tube?

DR. JOHN A. LIGHTY, Pittsburgh, Pa: The reader seems to have covered the ground so well that there is little more to say, except to give personal experience. My experience coincides with that of Dr. Aaron in that the two tests do not always agree. I may quite frequently get a certain result with the Ewald test and find a slightly different result with the Rehfuß. I have, therefore, abandoned the Rehfuß test as a routine measure. In cases in which the psychic side enters as a large factor, I prefer the Ewald method for obvious reasons. If a nervous patient has to spend two or three hours with a tube in her mouth acting as a foreign body in the stomach also, you might as well quit if you are expecting to get a normal function of the stomach at that time. I prefer to take these patients without any ceremony, give very little explanation, have them take the test meal and not anticipate what is about to happen, and promptly at the end of the hour or of fifty minutes introduce the tube and withdraw the contents. There are certain conditions, however, in which I depend very largely upon the Rehfuß. Where the single test shows an apparent achylia, I insist upon doing the Rehfuß to see whether this absence of hydrochloric acid extends through the whole digestive cycle. I believe with proper interpretation and care such a procedure will not mislead one.

I also use it in cases in which I am suspicious of certain pathology in the mediastinum which might interfere with the introduction of the Ewald tube. The swallowing of the Rehfuß tube is a voluntary act and it is entirely in the hands of the patient. He cannot do himself any harm and I feel safer in the use of this than I do in pushing violently the Ewald tube into the stomach.

I have not used the stylet, as suggested by Dr. Aaron, but believe in certain cases this procedure would be very satisfactory.

I might suggest it would add to the strength of Dr. Aaron's paper if he would show that his diagnoses have been proved by autopsy, or at operation, or probably with X-ray.

Finally, I will say that the Rehfuß test is a very cumbersome one, a time consumer, and I believe we are wasting a great deal of time if we insist upon using this test routinely when the Ewald under proper circumstances and precautions gives such valuable information.

DR. A. H. AARON, Buffalo: We did not have the patients swallow the tube, we passed it very carefully, and slowly. This, we believe, accounts for the little trauma caused which would produce bleeding, and interfere with the accuracy of our results.

DR. E. H. GAITHER, Baltimore: We are in entire accord with the views of Dr. Aaron in his splendid paper.

Last year in a presentation before this society we called your attention to the futility in the great majority of cases, of applying fractional gastric analysis relative to obtaining information which would prove to be of true value from a diagnostic standpoint, and at the same time presented our reasons therefor.

Naturally it is most reassuring to hear Dr. Aaron corroborate our views in such a thorough manner this morning.

DR. JACOB KAUFMANN, New York City: I think that Dr. Aaron's paper is a most valuable contribution to the question of studying the gastric secretion. It shows conclusively that the Rehfuß method is not reliable in deciding the status of a patient. It is of value in the study of certain features of gastric secretion and will be employed for such studies, but for practical bedside purposes the older method of the single test meal yields more reliable information. I would not be willing to discard the older method of examination of gastric contents, because the fractional examination has shown such varying figures for acids. I do not think that we should lay stress on finding slight differences. Whether total acidity reads 40, or 70 or 80, does not count. We must get marked differences, such as achylia, subacidity or a pronounced increase. I further think that we should look more for products of digestion and differentiate degrees of peptic activity of gastric secretions.

Some time ago I read a paper here on the lack of mucus in gastric contents. When you find very little mucus it shows that the mucus has been digested. In a case of achylia, you find on microscopical examination gluten well preserved, while in cases of strong peptic secretion it is entirely missing. Where you find the starch isolated and gluten and mucus completely digested you can diagnose a strongly digestive gastric secretion. I am able to diagnose the peptic strength of gastric secretion by microscopical examination. The microscopic picture of achylia is very typical, and so is that of hyper-secretion. These things are not without value, and I would not discard examinations of gastric contents altogether. You will find that a person, as a rule, has the same type of secretion. Some go without secretion; and there are others with a low secretion, and others with pronounced secretion. Others, again, as Dr. Hemmelter first pointed out, will at one time show little secretion, and at others an increased secretion. It is worth knowing such a state of heterochylia because it shows an unstable vegetative nervous apparatus. People who are inclined to have a state of increased irritation in their vegetative apparatus are more inclined to get ulceration, but we must not take such signs as evidence of ulceration. So I think, after all, that if Dr. Aaron's paper does away with the routine employment of a method which has proved of doubtful value for practical purposes, that it will not do away with examinations of gastric contents altogether.

DR. THOS. R. BROWN, Baltimore: I enjoyed this paper because it points out the fact that most students have failed, to a great extent, to recognize for many years, and that is that because a method is propagated it must necessarily, be a satisfactory method. We have been preaching in our clinic for a great many years and urging the unsatisfactory results of the fractional method which Dr. Gaither showed in his paper last year. When you realize the fact that we are supposed to lead thought in regard to digestive diseases in this country, you will admit that we have a great deal on our conscience. This is not a society where methods should be changed or new methods adopted and retained without serious investigation. That certain methods are followed and certain views held by a number of men, and good men at that, is due to the fact that we have a tendency to follow any methods, because they are new, and not analyze the underlying physiology.

After all, this method is as old as the hills. Many years ago, the French using an ordinary stomach tube, introduced at definite intervals rather than a permanent tube, attempted to evaluate the secretory curve, obtained in this way. If you read these books you will find the same types of curves. That was all tested out, and discarded because it was not sound.

The point of the paper which is fundamental is that with the stratification of gastric contents, it is impossible to tell, when you get a small amount, what you are dealing with; and that the total evacuation of the stomach contents at a definite time really tells us more, in most cases than the fractional method. There are so many sources of error that only in a very rough way will secretory readings be of value. We are all sinners, to greater or less extent. If we had utilized the amount of time that has been devoted to the fractional method in the last five or six years, how much work could we not have produced along better lines, and how much valuable time is wasted by thousands of patients, sitting for hours with tubes in their mouths. At the end we have learned nothing that could not have been told by the brain of the doctor, a fasting stomach content and one gastric meal. You can only tell that the patient has a tendency to low acidity, to high acidity, to normal acidity or to no acidity.

All of the experiences of the last few years teaches us that we should not support anything because it gives us curves and smacks of a scientific method. A method is not scientific because of its complexity but because it is based on a sound scientific foundation. We have a duty to perform, to persuade the body politic of physicians that we are capable men, doing work on physiological grounds, and trying to base our studies on a firm physiological foundation. If they are so based, they can always stand scrutiny. We have been rather, and rightly, open to criticism in the past for supporting methods that live for a while and are then cast aside.

DR. CLEMENT R. JONES, Pittsburgh, Pa.: I just want to call attention to what I consider a minor point in doing gastric analysis, and that is to the use of fresh breads and stale bread, partly baked bread and well baked bread, dry bread and moist bread, and all the varieties of bread employed in making a gastric analysis. Gorham, in his work, has been using shredded wheat, and I have been using shredded wheat for some time. The bread may contain chemicals that would influence the character of the gastric secretion and if we develop a more stable type of test breakfast, I think that we shall get more constant results in our findings. I am entirely in accord with the essayist on this subject and with the remarks of Dr. Brown. My experience in making examinations of the gastric contents with various objects in view has led me to take on different days, under as nearly the same circumstances and the same conditions of the patient as possible, a meal at half an hour, one at an hour, and one at an hour and a half, with the same large tube. This method of procedure gives a better idea of the gastric conditions than I could obtain with the Rehfuß or the single test breakfast.

DR. JOHN C. HEMMETER, Baltimore: I wish to congratulate the society upon the reading of a paper that gives every evidence of very broad experience and conservative judgment. I have never read a paper on this fractional analysis of the gastric contents or spoken on it, but I have accumulated a very large amount of material in private and hospital practice on the subject. The matter of regurgita-

tion of bile, I wish to speak of first. Boldyreff does not consider this to be normal. I have had three students living with me for nearly a year; and every morning they wash their stomachs out at times they take test meals for experimental purpose. One student had bile in the stomach twice in twelve months; the others have never had it. The idea that gastric juice is an ever constant secretion is physiologically and anatomically impossible. The construction of the human stomach, as brought out by F. P. Mall in a beautiful paper, shows that only the middle part of the stomach contains the oxyntic cells, which are the laboratory for the production of hydrochloric acid. The opinion that all parts of the stomach can produce equally the same product is anatomically unfounded. I produced successfully, last summer, two operative isolated pouches at the pyloric end of the stomach and the secretion at the pyloric end is alkaline.

DR. A. H. AARON, Buffalo (closing): From known physiological facts we are able to state that the gastric secretion varies according to the location from which the specimen is secured, varying whether it is obtained from the fundic, prepyloric, or pyloric region of the stomach.

Dr. Franklin W. White has observed the duodenal tube in situ by means of the fluoroscope, and found that the tip sought the lowest level of the stomach, and remained there.

DR. A. A. JONES: Was it near the pylorus?

DR. AARON: It was located approximately opposite the spine.

The bread used was obtained from the same manufacturer, and was twenty-four hours old.

Regarding the swallowing of the tube, we readily recall the older test to determine whether an individual was of the neurotic type or not, namely passing a sterile swab over the conjunctiva or using the tongue depressor to elicit the pharyngeal reflex. A failure to respond to either of these procedures leads one to conclude that the individual is subnormal, while if there is any exaggerated response we conclude a hypersensitiveness of the nervous system. It is the same in regard to swallowing the tube, some take it readily, others less easily, and a number with great difficulty. This factor often causes nausea, retching, and the associated duodenal regurgitation that occurs coincident with this.

Dr. M. E. Rehfuess has stated that 40 per cent of his cases demonstrated duodenal regurgitation, which, in his opinion, is a marked factor in altering the character of the gastric secretion.

In regard to Dr. Lichty, I might say that all cases were studied under approved methods as thoroughly as possible, and that cases of gall-bladder and appendix diseases were proved by surgical intervention.

In conclusion we might state that the secretory values are of considerable worth in substantiating a diagnosis such as pernicious anemia, where there is a constant achylia. Subacid or achylia figures have more diagnostic significance than the hyperacid values.

We made no attempt whatsoever to discuss motor gastric phenomena in this paper.

## H-ION CONCENTRATION METHOD OF GASTRIC ANALYSIS

JOHN H. KING AND SAMUEL HALPERN

*Baltimore, Maryland*

Since Prout in 1824 showed that HCl was present in gastric contents and Szabo in 1877 devised a method for titrating the HCl, a considerable amount of progress has been made in the understanding of the nature of acidity. It will perhaps not be amiss to consider briefly the subject of acidity in a general way, before taking up the specific discussion of gastric acidity.

### ACIDS

Acids on dissociation give hydrogen ions as the cations regardless of the kind of acid. Bases on dissociation give hydroxyl ions as the anions regardless of the kind of base. A solution is acid when there are more  $H^+$  than  $OH^-$  in solution and alkaline when the  $OH^-$  are more numerous. If both are present in equal amount the solution is neutral. Water itself dissociates into  $H^+$  and  $OH^-$  and partakes therefore of the nature of acid and base. These ions are present in equal amount and so water is neutral. The equilibrium which has been established means that water dissociates into its ions at the same speed at which the ions unite to form water, which is an example of the law of mass action. The speed of the reaction is proportional to the concentration of the interacting substances. When equilibrium has been established the speed in either direction is the same.

### EXPRESSION OF ACIDITY

In determining the acidity of solutions one measures the amount of hydrogen ions per liter or the hydrogen ion concentration per liter and this is expressed in terms of a fraction of a gram of hydrogen ions per liter. The hydrogen-ion concentration of pure water is  $10^{-7}$ . A solution is acid when the ( $H_+$ ) is greater than the  $10^{-7}$ , or alkaline when the ( $H_+$ ) is less than  $10^{-7}$ . As the hydrogen ions increase in number the hydroxyl ions must decrease in number since their product equals  $10^{-1}$ . The more acid solutions are represented by the smaller pH values and every pH represents a tenth-fold increase in acidity.

The hydrogen ion is the portion of any acid which gives to it acid character, while the hydrogen ion concentration determines the degree of activity of the acid. Those acids which are slightly dissociated, as carbonic and acetic are the weak acids whereas those highly dissociated are the strong acids. In the same concentration HCl has 50 times as many ions as acetic acid or 1000 times as many as carbonic acid and is proportionally active. The salts of weak acids are almost completely dissociated, yielding a large number of the same anions as those of the acid. By the mass law, the ionization of the acid is repressed and in amount proportional to the amount of salt present.

#### DETERMINATION OF ACIDITY

The strength of acids is subject not only to theoretical consideration but also to experimental verification. The two principal methods in use for determining acidity are (1) the electrometric and (2) the colorimetric.

1. *The electrometric method.* This is based upon the principle originated by Nernst, that is if one connects two solutions of the same substance in different concentrations, a current is produced which is proportional to the difference in concentration. Consequently if one knows the concentration of one solution and the voltage, one can calculate the concentration of the other. The principle has been applied to determine the hydrogen ion concentration of any solution. The method requires elaborate apparatus and is difficult and time consuming. Because of its accuracy, new problems necessitate its use.

2. *The colorimetric method.* Based upon measurements made by the electrometric method, Sorensen devised the colorimetric method. Here indicators are used to measure the hydrogen ion concentration. There is a zone of hydrogen ion concentration in which each indicator develops its characteristic color changes. The method requires a series of buffer solutions of which the pH is accurately known. The proper indicator is added to equal portions of the standard and the sample. The colors are then compared and the pH of the sample is taken as the pH of the standard to which it is closest in color. This method is easy and rapid and gives values which are accurate for all clinical purposes. The essentials for this method are (1) a set of standard buffer solutions and (2) indicator.

### *Buffers*

Buffer means a solution which will hold the reaction constant or nearly so, when acid or alkali is added to it. Two types of buffer action are clearly understood.

1. If a weak acid is present in solution with its salt the addition of acid or alkali does not cause a great change in the reaction, for the acid is only slightly dissociated while the salt is almost completely so. The pH is dependent upon the proportion of the acid to the salt. Therefore until all the acid has been converted into the salt, the addition of strong alkali will not make the solution very alkaline, or until all the salt has been converted into acid, the addition of a strong acid will not make the solution very acid.

2. A second type of buffer reaction is similar to the first, but still more stable. If on addition of an acid or an alkali to a solution, a compound is formed which in itself is slightly dissociated, there will be a very small change in the H-ion concentration. Protein has this property of forming compounds with either acids or bases yielding slightly dissociated compounds. In general di-basic or poly-basic acids with one or more  $H^+$  neutralized are in this category. Other substances act as buffers through their ability to absorb or adsorb acids or alkalis. Charcoal and colloids probably fall into this class. The question has often arisen as to which buffer is the best. Koppel and Spiro have shown that all buffers are equally good but that each is strongest in a certain zone. They demonstrated that the maximum buffer value occurs at the ionization constant, or when the acid and salt are present in equal amounts. Practically the buffer solution is selected in which the ionization constant is nearest the desired pH.

### *Indicator*

So many indicators are available that considerable judgment must be used in selecting those most suited to gastric analysis. A great many substances show change in color in acid and alkaline solutions. If strong acids and strong bases are titrated almost any of the common indicators can be used. That is because their "end point" or color change is within the pH produced by a single drop of strong acid or alkali. When protein or other buffers are present, the indicators give different results. Indicators which give accurate values in pure solutions may be at fault in biological fluids, because of the presence in them of protein or salt. To determine the pH of gastric contents one

must first define the necessary range of pH. At pH 3.0, 100 cc. of contents equals only 1 cc. of  $N/10$  free HCl. Large amounts of organic acids or acid protein may be present without increasing the pH. Hence pH 3.0 should be taken as the least acid part for the study of free HCl. If 70 cc. of  $N/10$  free HCl are present in 100 cc., the reaction is pH 1.2. Several indicators that cover this range have been recommended for determining the pH of biological fluids. Thymol-sulphon-phthalein studied by Clark and Lubs is the only indicator which meets the requirements for determining the pH of the gastric contents.

#### THE ACIDITY OF STOMACH CONTENTS

From a consideration of the pH of solutions and the strength of acids the nature of acidity of stomach contents becomes clear. If we add HCl to stomach contents the hydrogen-ion concentration will not reach pH 3.0 until all the protein and all the organic acids have been freed from their combination with protein by HCl. For this reason the sum of all the acid factors is called total acidity, of which the acidity from neutrality to pH 3.0, is combined acidity and from pH 3.0 to pH 1.2 is free HCl.

*Free HCl.* At pH 3.0 100 cc. of stomach contents represents 1 cc. of  $N/10$  HCl, not combined with protein. This is so, regardless of the presence of large amounts of free organic acid. To give accurate titration values for free HCl, an indicator must be without protein or salt errors and have a sharp end point at pH 3.0.

*Total acidity.* The total acidity of stomach contents is the amount of free acid plus the acid combined with protein and other buffers. It is usually determined by titration with phenol-phthalein.

#### SIGNIFICANCE OF pH VALUES IN GASTRIC CONTENTS

The importance of considering the acidity of stomach contents in terms of hydrogen ion concentration is brought forth by analysis of recent literature on peptic digestion. Sorensen in his work on enzymes showed that the acidity of the medium was as important a factor as temperature in enzyme action. Only within certain narrow limits of reaction is pepsin active. The optimum reaction is approximately pH 1.65 for the action of pepsin on acid egg albumen. This is in closer agreement with the figures of Michaelis and Davidson who recorded the optimum for peptic action on casein as pH 1.8. They show that this is due to the hydrogen ion concentration and not to any

specific qualities of HCl, as they were able to duplicate these values with another mineral acid  $\text{HNO}_3$  and two very strong organic acids, tartaric and oxalic. Christiansen finds the optimum for human pepsin to be pH 1.55. If the amount of acid is greater or less than this, the amount of protein digestion becomes smaller. Concentrations of acid greater than pH 1.3 permanently injure and finally destroy the enzyme. If the reaction is not more acid than pH 3.0 peptic action is very poor and at pH 4.0 is almost lacking. The fact that pepsin requires a solution of definite pH for its action is supplemented by our knowledge of the reaction of gastric juice. Foa and Tangyl working with pure gastric juice determines the pH by the electro-metric method and found it to be pH 1.3. Frankel, Michaelis and Davidson and Christiansen found that the normal of the contents after an Ewald's meal lay between pH 1.6 and 1.5. So the normal hydrogen ion concentration of gastric juice makes an ideal medium for the optimum action of pepsin.

#### THE COLORIMETRIC DETERMINATION OF FREE HCl

There are two clinical methods based upon the colorimetric: (1) Muller's, (2) Michaelis and Davidson's.

*Muller's method* involves the use of standard solutions containing known amounts of HCl and the indicator tropaeolin 00. The stomach contents and the standards are put in tubes of the same diameter and same amount of indicator is added to each. One compares the color of the contents with the standard which most nearly matches it and the amount of HCl is estimated. Since the indicator solutions fade, Muller has devised a color chart to replace the standard.

*Michaelis and Davidson's method* is a rough or orientation procedure. By the use of seven properly selected indicators, the range of hydrogen ion concentration of any stomach content from alkalinity to hyperacidity can be approximately determined.

Christiansen showed that in the presence of the protein as in stomach contents, tropaeolin 00 gives results that are not accurate, hence Muller's method must be rejected.

*Author's method.* Based upon the colorimetric method for measuring hydrogen ion concentration.

## SOLUTIONS AND APPARATUS REQUIRED

1. Standard solutions are made according to the method of Clark and Lubs from pH 1.3 to 3.0. With the exception of the least acid (pH 3.0) which is tenth normal acetic acid, they are mixtures of HCl and KCl. As many standards can be made as desired.

*Indicator.* The range of acidity of stomach contents for free HCl is pH 1.2 to 3.0. From the indicators which cover this range thymol-sulphon-phthalein was chosen. This indicator exactly covers the range, is brilliant, permanent and free from errors due to protein and

TABLE 1  
*Standards and values in terms of pH and N/10 HCl*

| pH  | N/10 HCl IN 100 CC. OF CONTENTS |
|-----|---------------------------------|
|     | cc.                             |
| 1.3 | 54                              |
| 1.4 | 44                              |
| 1.5 | 35                              |
| 1.6 | 27                              |
| 1.7 | 22                              |
| 1.8 | 17                              |
| 1.9 | 13                              |
| 2.0 | 10                              |
| 2.2 | 6                               |
| 2.4 | 4                               |
| 2.6 | 2.5                             |
| 3.0 | 1.0                             |

salts. Clark and Lubs mention its applicability to stomach contents. Color of indicator is wine red at pH 1.2, orange at pH 2.0 and yellow at pH 3.0. The colors will last for months if the color stands when not in use are kept in a closed box at room temperature. A 0.1 per cent alcoholic solution is used. Inasmuch as the color of thymol-sulphon-phthalein changes most marked at points corresponding to normal or slightly diminished gastric acidity, it is most useful in the most important range. Strongly acid solution can be brought to this range by simply diluting.

*Color comparator, Hynson and Wescott's apparatus*

*Material.* The gastric contents were removed one hour after an Ewald test meal of 250 cc. of water and 25 grams of bread. The contents were either filtered or unfiltered. In the latter case, they were

centrifuged at high speed for ten minutes and the supernatant fluid drawn off. In this way considerably more material for analysis was obtained.

*Method.* Take 2 cc. of filtered or centrifuged contents recovered after an Ewald test meal and transfer to a glass container as provided in the Hynson and Wescott Apparatus. Add 1 drop of the indicator solution for each cubic centimeter of gastric content used. Place in the comparator, and select the standards which most closely match until the closest possible match is made. The markings on the standard color ampule will give the hydrochloric acid per 100 cc. of gastric juice. (For more minute details see original article, Shohl and King, Johns Hopkins Hospital Bulletin, vol. xxi, no. 351, May, 1920.)

The results can be read directly in terms of pH, normality, or in cubic centimeters of N/10 HCl. It is not necessary to use the amounts here given. The only requisites are (1) the indicator must be in the same proportions in the standards and sample, (2) the tubes containing the standards and sample must be the same diameter, (3) no water must be added unless the amount is known and the necessary calculations made.

*Checks.* (1) By titration with N/10 alkali using Topfers indicator. (2) By titration with N/10 alkali using thymol-sulphon-phthalein as an indicator. (3) By dilution. (4) By the electrometric method.

#### COMBINED ACIDITY AND BUFFER VALUE

The combined acid is that portion which remains after the free HCl has been neutralized and consists of (1) the combined HCl, (2) organic acids and (3) acid salts. Experience has shown that the combined HCl is practically the same as the combined acid, for organic acids and acid salts play a very small part in gastric acidity. In cases in which no free HCl occurs the combined acid and the total acidity are identical. Such contents are said to show an acid deficit, represented by the amount of HCl which must be added to the sample until free HCl is just present. This value indicates the amount of acid which the stomach should secrete, or which, if it fails to secrete must be added before gastric digestion can progress. The sum of the total acidity and the acid deficit in such cases measures the power of the stomach contents to combine with acid. This value is comparable to the combined acids in cases showing free HCl.

*Buffer value.* Buffer solutions are those to which acid or alkali can be added without causing a marked change in the reaction. They

possess this property because they are either weak acids which in addition of an alkali do not become alkaline until all the acid has been neutralized, or they are salts of weak acids which in addition of acids do not become acid until the weak acid has been freed from its salt. In stomach contents the protein acts as a buffer. It can combine with acid or base and give nearly neutral compounds. The amount of acid or alkali which can be added is called the buffer value. The amount of acid or alkali necessary to change the reaction from one hydrogen ion concentration to another is the buffer value between these acidities. The total buffer value is the amount necessary to change the reaction from pH 3.0 (no free HCl) to pH 11.0 free alkali.

F. Volhard (1903) was the first to show that in stomach contents titrations with phenol-phthalein give higher values than those with litmus, because it is an indicator for alkaline solutions and more alkali must be added to reach the end point. He also showed that this is due to the buffer action of the protein of the contents. What is really determined, is the buffer value to the end points of the indicators.

#### FACTORS AFFECTING BUFFER VALUE

##### *1. Concentration*

In general one may say that the buffer value is not related to the hydrogen ion concentration, but the hydrogen ion concentration to the buffer value. The more concentrated the buffer solutions, the greater is the amount of acid necessary to change the solution to a desired pH.

##### *2. Hydrolysis of protein*

As peptic digestion progresses the proteins become hydrolyzed or split into polypeptids or peptones, thereby producing more free alkaline groups. These combine with more HCl, thus reducing the hydrogen ion concentration and free HCl and increasing the buffer value.

##### *3. Other factors*

Extraneous buffers, among which may be carbonates regurgitated from the duodenum, blood and foreign protein or phosphates, also increase the buffer value of the contents.

*Method.* To find the buffer value of a sample of stomach contents, one must determine the amount of acid or alkali necessary to change its reaction from pH 3.0 (no free HCl) to pH 9.6 (near the point of free alkali).

*Indicator.* Thymol-sulphon-phthalein is the indicator used. It has an acid color zone red (pH 1.2 to 3.0); a neutral color zone yellow (pH 3.0 to 8.0); and an alkaline color zone blue (pH 8.0 to 9.6). It gives nearly the total buffer value from free HCl to free alkali.

#### PROCEDURE

##### *A. When free HCl is present*

Add 1 drop of the indicator (0.1 per cent thymol-sulphon-phthalein) for each cubic centimeter of stomach contents. Titrate with N/10 NaOH free from carbonate to the full blue color of the indicator. Subtract the value of the free HCl found by the colorimetric method from the titration value.

##### *B. When no free HCl is present*

*a.* Determine first the acid deficit. Add 1 cc. of N/20 HCl to 2 cc. of the gastric content and 1 drop of the indicator for each cubic centimeter of fluid in the ampule. Read the value of the free HCl by the colorimetric method. Multiply this value by the number of cubic centimeters present (stomach contents plus acid) and subtract the product from 50. Divide this result by the number of cubic centimeters of stomach content used and the result will be the value of the acid deficit.

*b.* To the acid deficit obtained thus, add the alkaline titration value. To obtain this titrate 2 cc. of the gastric content with N/10 NaOH free from carbonate, until the full blue color is present. This gives the total acidity. The sum of the acid deficit and total acidity give the buffer value. (For more detailed description see original article, Shohl and King, Johns Hopkins Hospital Bulletin, vol. xxi, no. 351, May, 1920, and special circular of Hynson and Westcott.)

#### DISCUSSION

The buffer value and the combined acid value, although almost equal in numerical terms, differ greatly in significance. The combined acid value signifies the amount of HCl combined with the protein. The buffer value represents the amount of acid or base required to bring about a definite change in reaction. This method determines neither the acid production, nor the acid value of the stomach contents, nor the amount of alkali necessary to bring the contents to the alkalinity of the blood. By measuring also the acidity

of the food, however, these three factors can be determined from the buffer value to certain end points—to the reaction of the food, for the acid secretion, to pH 7.0 for the acid value of the contents, and to pH 7.4 for the amount of alkali necessary to bring the contents to the reaction of the blood.

#### EXPERIMENTAL

The methods which have been described were employed in the analysis of a series of clinical cases. The material consisted mostly of cases admitted to the gastro-intestinal clinic of the Johns Hopkins

TABLE 2

| GROUP                                      | CASES | AVERAGE<br>BUFFER | AVERAGE<br>pH |
|--|-------|-------------------|---------------|
| Splanchnoptosis . . . . .                  | 18    | 24.4              | 1.65          |
| Chronic appendicitis . . . . .             | 15    | 27.8              | 2.03          |
| Chronic gastritis . . . . .                | 5     | 23.2              | 2.27          |
| Gall-bladder disease . . . . .             | 11    | 19.1              | 2.3           |
| Gastric neuroses . . . . .                 | 8     | 24.3              | 1.99          |
| Post-operative adhesions . . . . .         | 2     | 25.0              | 2.02          |
| Syphilis . . . . .                         | 5     | 13.6              | 1.51          |
| Tuberculosis . . . . .                     | 2     | 30.5              | 1.55          |
| Hyperthyroidism . . . . .                  | 1     | 4.0               | 2.0           |
| Genito-urinary diseases . . . . .          | 1     | 31.0              | 1.49          |
| Epilepsy . . . . .                         | 1     | 16.0              | 1.49          |
| Obesity . . . . .                          | 1     | 21.4              | 1.8           |
| Cancer of intestines . . . . .             | 2     | 17.0              | 2.4           |
| Ulcers (duodenal) . . . . .                | 2     | 39.5              | 1.8           |
| Respiratory diseases (influenza) . . . . . | 1     | 32.0              | 1.78          |
| Cardio-vascular diseases . . . . .         | 2     | 24.5              | 2.19          |
|  | 77    | 23.3              | 1.89          |

Hospital Dispensary and from the public and private wards of the Johns Hopkins Hospital. The series was comprised of groups of cases showing free HCl and others showing no free HCl in the gastric content removed one hour after an Ewald test breakfast.

1. In the group showing free HCl there were 77 cases. In each the free HCl, total acidity, buffer value and the hydrogen ion concentration were determined. The findings will be considered in groups. The values of the various factors which were determined varied greatly in each group, so that the average values for a group were considered as giving a better idea of the relations in each group. The

average buffer values and hydrogen ion concentrations were the special points of interest. The groups are shown in table 2.

In analyzing the groups it will be found regarding *the hydrogen ion concentration* that the highest average value was found in genito-urinary diseases and epilepsy, and the lowest in cancer. If the

TABLE 3  
*Hydrogen-ion concentrations of gastric content*

| LOW pH 3.0 TO 2.2<br>(LOW FREE HCl)                                       | MEDIUM pH 2.0 TO 1.7<br>(AVERAGE FREE HCl)   | HIGH pH 1.6 TO 1.3<br>(HIGH FREE HCl)  |
|---|--|--|
| Cancer of intestines<br>Gall-bladder diseases<br>Cardio-vascular diseases | Chronic appendicitis<br>Chronic gastritis<br>Gastric neurosis<br>Hyperthyroidism<br>Obesity<br>Duodenal ulcers<br>Epidemic influenza<br>Post-operative abdominal adhesions | Splanchnoptosis<br>Genito-urinary diseases<br>Tuberculosis<br>Syphilis<br>Epilepsy |

TABLE 4  
*Buffer values of gastric contents showing free HCl*

| HIGH BUFFER (28 TO 39)   | MEDIUM BUFFER (16 TO 28)   | LOW BUFFER (4 TO 16)                    |
|--|--|---|
| Genito-urinary diseases<br>Tuberculosis<br>Duodenal ulcers<br>Epidemic influenza | Gall-bladder diseases<br>Gastric neurosis<br>Post-operative abdominal adhesions<br>Splanchnoptosis<br>Chronic appendicitis<br>Obesity<br>Cancer of intestines<br>Cardio-vascular Lesions | Hyperthyroidism<br>Epilepsy<br>Syphilis |

groups are arranged according to low, medium, and high hydrogen ion concentrations, the results may be expressed as in table 3.

2. *The buffer values.* The lowest figures were found in hyperthyroidism, and the highest in duodenal ulcers. The buffer values varied from 4.0 the lowest to 39.5 the highest. If the groups are arranged according to low, medium and high buffer values the results may be expressed as in table 4.

*Remarks.* The majority of the disease groups show medium values for the hydrogen ion concentration and buffer.

In general, it may be said that the buffer value is not related to the hydrogen ion concentration but the hydrogen ion concentration to the buffer, since the more concentrated the buffer solution the greater is the amount of acid necessary to change the solution to a desired hydrogen ion concentration.

The average buffer value for the entire groups is 23.3 which will be of interest when the cases showing no free HCl are considered.

In the cases showing no free HCl, 35 cases were analyzed. The acid deficit, total acidity, buffer values, and combined HCl (or the amount of HCl secreted) were determined. The cases of this series can be divided into groups, containing examples of practically every clinical form of achylia gastrica.

A simple clinical classification of the achylia is the following one:

1. *Achylia, associated with systemic disorders; no pathological lesion apparent in the stomach*
  - a. Hyperthyroidism
  - b. Pernicious anemias
  - c. Diabetes
  - d. Splanchnoptosis
2. *Achylia associated with organic lesions in the stomach*
  - a. Cancer
  - b. Chronic gastritis
3. *Nervous achylia*

The clinical cases investigated contained cases of each of these groups.

The acid deficit can be used to determine the amount of any bound HCl present, that is whether any HCl at all has been secreted and if so how much. To determine this, the maximal quantity of HCl that the protein and the amino acids present can bind must be known. If the HCl deficit is subtracted from this, the amount of HCl that has actually been secreted and bound is found. The maximal amount of HCl that can be bound by the stomach contents depends upon the amount of proteins and the other bases present. It has been shown that the ordinary Ewald test breakfast contains an approximately constant amount of these substances, so that after a test breakfast a fairly constant amount of HCl is bound. In 100 cc. of filtered stomach contents this corresponds to 20 cc. N/10 HCl; that is to say, if no HCl were secreted by the stomach, it would be necessary to add to 100 cc.

of filtered gastric juice, 20 cc. of N/10 HCl in order to satisfy all the proteins and bases present. The value 20 then represents the maximal HCl deficit after a test breakfast, as far as the protein in the test breakfast itself is concerned. If therefore the HCl deficit is subtracted from 20, the value of the combined HCl present is found. This was determined in our groups of cases. The groups shown in table 5 were investigated.

TABLE 5  
*Achylia*

| GROUPS                               | CASES | AVER-<br>AGE<br>ACID<br>DEFICIT | TOTAL<br>ACIDITY | AVER-<br>AGE<br>BUFFER | AVER-<br>AGE<br>COM-<br>BINED<br>HCl |
|--------------------------------------|-------|---------------------------------|------------------|------------------------|--------------------------------------|
| Chronic appendicitis . . . . .       | 4     | 8.6                             | 16.0             | 24.6                   | 11.4                                 |
| Splanchnoptosis . . . . .            | 8     | 10.3                            | 13.1             | 23.4                   | 9.7                                  |
| Gastric neuroses . . . . .           | 3     | 8.6                             | 18.0             | 26.6                   | 11.4                                 |
| Colitis . . . . .                    | 1     | 12.0                            | 9.5              | 21.5                   | 8.0                                  |
| Gall-bladder disease . . . . .       | 4     | 11.5                            | 11.3             | 22.8                   | 8.5                                  |
| Cancer of stomach . . . . .          | 2     | 7.0                             | 16.8             | 28.5                   | 13.0                                 |
| Chronic gastritis . . . . .          | 2     | 9.5                             | 18.0             | 27.5                   | 10.5                                 |
| Pernicious anemias . . . . .         | 5     | 16.9                            | 5.5              | 22.4                   | 3.1                                  |
| Diabetes insipidus . . . . .         | 1     | 9.0                             | 7.5              | 16.5                   | 11.0                                 |
| Syphilis . . . . .                   | 1     | 13.0                            | 18.0             | 31.0                   | 7.0                                  |
| Hyperthyroidism . . . . .            | 1     | 2.5                             | 17.0             | 19.5                   | 17.5                                 |
| Tuberculosis . . . . .               | 2     | 16.0                            | 5.8              | 21.8                   | 4.0                                  |
| Cancer of head of pancreas . . . . . | 1     | 6.0                             | 36.0             | 42.0                   | 14.0                                 |
|                                      | 35    |                                 |                  | 25.1                   |                                      |

In analyzing the groups it will be seen regarding the *acid deficit* that the lowest average deficit occurred in hyperthyroidism and the highest in pernicious anemia. The acid deficit varied from 2.5 to 16.9. If the groups are arranged according to low, medium and high acid deficits, the results may be expressed in table 6.

The total acidities varied from 5.1 to 36. The lowest average value was found in pernicious anemia and the highest in cancer of the head of the pancreas. If the groups are arranged according to low, medium and high total acidities, the results may be expressed in table 7.

In general where the acid deficit is high, the total acidity is low, and vice versa.

The buffer values are more constant and do not show very wide variations, the lowest being 16.5 in diabetes insipidus, the highest, 42, in cancer of the head of the pancreas.

If the buffer values are arranged according to low, medium and high values the results can be expressed in table 8.

TABLE 6  
*Acid deficit*

| LOW (2 TO 7)    | MEDIUM (7 TO 12)   | HIGH (12 TO 16)   |
|-----------------|--|---|
| Hyperthyroidism | Chronic appendicitis<br>Gastric neuroses<br>Gall-bladder diseases<br>Cancer<br>Chronic gastritis<br>Diabetes insipidus | Splanchnoptosis<br>Colitis<br>Pernicious anemia<br>Syphilis<br>Tuberculosis |

TABLE 7  
*Total acidities*

| LOW (5 TO 15)   | MEDIUM (15 TO 25)  | HIGH (25 TO 36)            |
|---|--|----------------------------|
| Splanchnoptosis<br>Colitis<br>Gall-bladder diseases<br>Cancer of stomach<br>Pernicious anemia<br>Diabetes insipidus | Chronic appendicitis<br>Gastric neuroses<br>Chronic gastritis<br>Syphilis<br>Hyperthyroidism<br>Tuberculosis | Cancer of head of pancreas |

TABLE 8  
*Buffer values*

| LOW (16 TO 24)   | MEDIUM (24 TO 32)   | HIGH (32 TO 42)    |
|--|---|--------------------|
| Splanchnoptosis<br>Colitis<br>Gall-bladder diseases<br>Cancer of stomach<br>Pernicious anemia<br>Diabetes insipidus<br>Hyperthyroidism<br>Tuberculosis | Chronic appendicitis<br>Gastric neuroses<br>Chronic gastritis<br>Syphilis | Cancer of pancreas |

*Remarks.* The average buffer value is 25.1 while that for the cases with free HCl is 23.3. These figures are quite close in agreement for a large number of cases and show that the stomach has a fairly constant power of combining with acids under all conditions.

The average combined HCl, or the amount of acid actually secreted by the stomach in the different groups of achylia varies from 3.1 the lowest in pernicious anemia to 17.5 the highest in hyperthyroidism. If the average combined HCl values are arranged according to low, medium and high values, the results can be expressed in table 9.

TABLE 9  
*Combined HCl values*

| LOW (3 TO 8)      | MEDIUM (8 TO 13)     | HIGH (13 TO 18)            |
|-------------------|----------------------|----------------------------|
| Colitis           | Chronic appendicitis | Syphilis                   |
| Gall-bladder      | Splanchnoptosis      | Cancer of head of pancreas |
| Cancer of stomach | Gastric neurosis     |                            |
| Pernicious anemia | Chronic gastritis    |                            |
| Hyperthyroidism   | Diabetes insipidus   |                            |

TABLE 10

| VALUES       | LOW              | MEDIUM   | HIGH             |
|--------------|------------------|--|------------------|
| Acid deficit | Organic achylia  | Systemic achylia<br>Organic achylia<br>Nervous achylia | Systemic achylia |
| Buffer value | Systemic achylia | Systemic achylia<br>Organic achylia<br>Nervous achylia |                  |
| Combined HCl | Systemic achylia | Systemic achylia<br>Organic achylia<br>Nervous achylia |                  |

The amount of acid secreted in the different groups of achylia varies conversely as the acid deficit, that is if a small amount of acid is secreted the gastric contents will show a large acid deficit.

#### THE CLINICAL SIGNIFICANCE OF ACID DEFICIT AND BUFFER VALUES

The acid deficit, buffer values and combined HCl in this series of achylia show some interesting points.

a. The achylia associated with systemic disease show in general medium to high acid deficits, low to medium buffers, and low to medium combined HCl.

*b.* The achylas associated with organic lesions in the stomach show low to medium acid deficit, medium buffer, and medium to high combined HCl.

*c.* The nervous achylas show medium acid deficit, medium buffer and medium combined HCl (table 10).

From table 10 it will be seen that the most characteristic points are:

1. The organic achylas have a low acid deficit.

2. The systemic achylas have a high acid deficit, low buffer and low combined HCl.

3. The nervous achylas have average values for the acid deficit, buffer values and the combined HCl.

4. The contrast in the achylas of cancer of the stomach and pernicious anemia is interesting.

*a.* In cancer there is a low acid deficit and a high combined HCl.

*b.* In pernicious anemias, there is a high acid deficit and a low value for the combined HCl.

The cases were all given approximately a uniform test meal so that influence of this factor would be approximately constant in every case. That this is so is shown by the fact that the average buffer value is approximately the same in the cases showing free HCl and those showing no free HCl in the gastric content after the Ewald test meal.

#### DISCUSSION

DR. A. H. AARON, Buffalo: I appreciate very much the simple method of colorimetric determination of the hydrogen ion concentration of the gastric contents, originated and presented by Drs. King and Shohl. The introduction of this procedure will necessitate our becoming acquainted with a new set of acid values as standards.

DR. JOHN C. HEMMETER, Baltimore: I wish to emphasize a number of chemical features which, no doubt, are familiar to an adept like Dr. King, but are not so familiar to the members of the society in general. The definition of acidity may be attempted from two viewpoints: first, from the purely chemical viewpoint; and second, from the physico-chemical viewpoint. A chemist conceives an acid to be a combination in which hydrogen atoms can be replaced by metallic atoms. By the entrance of the metallic atoms, the acid character is neutralized. The degree of acidity can, therefore, be determined by the quantity of decinormal solution of alkaline hydroxide which is necessary to replace the hydrogen atoms. The physico-chemist, however, defines an acid as a chemical combination which, when dissolved in water, dissociates free hydrogen atoms ( $-H-$ ). According to the degree of this dissociation, we can distinguish between strong and weak acids. In a certain definite volume of a strong, and of a weak acid, the weak acid will be less dissociated than the strong acid.

The difference between these two viewpoints will become immediately clear when we determine the acidity of a  $N/32$  HCl solution and a  $N/32$  acetic acid solution. From the standpoint of the chemist, both these dilute normal solutions of acids should have the same acidity when taken in exactly the same quantity. From the standpoint of the chemist, 1 liter of  $N/32$  HCl and 1 liter of  $N/32$  acetic acid require the identical amount of decinormal solution of sodium hydroxide (NaOH) for their neutralization. But, for the physico-chemist, the acidity of  $N/32$  HCl is forty (40) times as great as  $N/32$  acetic acid solution; because the stronger acid dissociates more than the weaker acid. The degree of dissociation of 1 liter of HCl in a dilution of 32 liters of water is equal to 0.97; but that of the weaker acetic acid is equal to only 0.024.

Concerning the method of determining the acidity by titration, the chemist determines the *dissociated* H-ions, as well as the *combined* H-ions. The latter, which, in certain dilutions, are not yet dissociated, are called the "potential" H-ions; while those that are dissociated are called the "actual" H-ions. The physico-chemical chemist determines only the "actual" H-ions. The chemist determines both. For the physico-chemist, the urine generally reacts neutral or very faintly acid. There is no direct dependence between the H-ions acidity and acidity determined by titration.

DR. JOHN H. KING, Baltimore (closing): The method really determines the ionized acid that is present, the truly active acid. Whether the ionized acid will have any diagnostic significance different from the acid determined by the older methods, can not be said until a large series of cases have been analyzed. It can be objected that the gastric acidity as determined by the older methods does not meet the modern scientific standards. The hydrogen-ion concentration of the blood was not studied in our series.

The standards put up by Hynson, Westcott and Dunning are now believed to be very stable. If this method can give us an easy way of distinguishing an achylia in cancer from an achylia in pernicious anemia, it will be of great service, because these two diseases are often very difficult to diagnose especially in the early stages.

## LOCAL TREATMENT OF GASTRIC IRRITATION

CHARLES G. STOCKTON

*Buffalo, New York*

Many patients, from one cause or another, suffer from gastric irritation and irritability. Usually there is a definite physical cause, and most often this is gastritis, either general or localized. For instance, most of the distress in non-obstructive peptic ulcer is occasioned by the inflammatory process about the essential lesion. This is often true in carcinoma. There are other conspicuous causes of a local inflammatory state. An erosion near the pylorus with an overtone stomach may lead to a local gastritis, and the same may be said of erosions at the cardia. Gastritis of a more general type, with much irritability, occurs in stomachs congested from chronic impairment of the general circulation, or obstruction of the portal stream, as occurs in diseases of the liver. Then there are the cases which are strictly infective, secondary to long standing infection from the upper respiratory tract or mouth. One of the most frequent offenders in this way is pyorrhea alveolaris. This does not complete the list of etiologic factors, but suffices for the purpose.

The chief complaints of the patient are pain, distress, a sensation of pressure; also there are symptoms of motor irritability—eructation, regurgitation and sometimes pylorospasm or some other spastic state—all of which are somewhat dependent upon gastric irritation or irritability. Not only may the patient's symptoms be relieved and his confidence restored, but the case will progress better generally when this gastric irritability is alleviated.

Of course benefit may follow the use of lavage, or lavement of the gastric lining with some one of various medicaments. I wish to emphasize the importance of systematically using a local dressing to the irritable stomach. This certainly is not a new idea, but a very old one, and often employed unconsciously. Frequently, however, I find patients suffering needlessly because the simple practice of local treatment of the stomach does not sufficiently impress the clinician.

Let us consider a few of the agents that may be used conveniently as satisfactory topical applications to the gastric mucosa. One of the oldest is bismuth, which should be given in large doses (4 to 16 grams) when the stomach is empty. In fact local application should usually be made only when the stomach is empty or approximately so. Bismuth should be given stirred in water so that it may diffuse itself over the surface. Cerium oxalate is also of use and should be given in large doses (1 to 2 grams). Finely powdered charcoal, alone or mixed with chalk powder, in 2 to 4 gram doses, is best given in wafers, so that the dry powder may attach itself to the mucous membrane.

A remedy much used abroad, but comparatively neglected in this country, is kaolin. From 4 to 16, or even 30 grams of kaolin, stirred in water, taken on an empty stomach or half an hour before meals, will sometimes serve a good purpose.

The list of substances to be used as local dressings is not ended, but it is sufficiently long to convey my idea. A good deal might be said on modifications of the dressing. For instance, certain sedatives may be employed in combination with the agents above described. A favorite of mine is *pix carbonum*, which is rubbed up with kaolin (*pix carbonum* 1 part, kaolin 9 parts). Of this 1 or 2 grams is given in a capsule. It is surprising what relief will follow in a great number of cases from the local effect of *pix carbonum* in addition to kaolin.

Iodoform, in amounts small enough not to cause toxicity, blended with a powder and given in cachets or wafer papers, works admirably. Of course there are many other drugs which we may use for the purpose, such as tannic acid, chloretone, chloroform, hydrastis, and ichthyol.

Following a local dressing to an inflamed or irritable stomach, the appetite will be improved and more food may be taken with comparative comfort, thus raising general nutrition. The spasticity of the stomach will abate, and this goes far in mollifying symptoms. To establish a state of gastric comfort is to favor the general progress of the case. I have purposely omitted the consideration of various forms of local treatment, for instance medicated sprays. What I am trying to emphasize is the importance of the local dressing, or the *pansement gastrique* of the French, in the treatment of gastric irritation.

## DISCUSSION

DR. JOHN A. LICHTY, Pittsburgh, Pa.: I thought that probably Dr. Stockton's paper would treat, also, of those cases in which there is pure neurosis, and tell us all of his experience—what he did with those cases that have gastric distress. It may be that he has in mind cases in which there are really some disturbances of the mucous membrane, and in which local application is desirable. As far as his treatment of gastric irritation is concerned, I should like to agree with him definitely regarding the use of bismuth. I have learned in the last few years something about bismuth that may be new to the majority of the audience, and which I did not know before. A number of times, when we got to the point, the symptoms did not ameliorate and we were about to open the abdomen; and no X-ray was made, because the patients were in such distress that we did not wish to burden them. We gave 2 ounces of bismuth, and then it simply disappeared. Before, we had given 5 or 10 grains of bismuth every two hours, and had no results; but since we give  $\frac{1}{2}$  ounce of carbonate of bismuth in the morning, one a day, the results have been much better. The patients sometimes reject a little, but most of it remained in the stomach. That is what did good, and produced the effect that I think Dr. Stockton has in mind.

DR. ALLEN A. JONES: Was that in ulcer cases?

DR. LICHTY: No, in ordinary cases of irritation of the stomach. It is helpful, also, in cases of ulcer; but in these cases of neurosis with distress in the epigastrium, in which you have scarcely anything to build on but the psychic or neurotic element, I have found a great deal of benefit from the spinal douche—simply standing the patients by the wall and shooting at them hot and cold water, the Coach douche, so-called.

DR. CHARLES G. STOCKTON, Buffalo (closing): In answer to Dr. Lichty, I would say that I think it is sometimes a little difficult to determine, in neurotic cases, whether or not there is present, also, a cause of local irritation. When I am convinced that there is no local source of trouble, and that the symptoms depend on the neurosis, I treat the neurosis, and not the stomach; but I almost invariably, in these cases, use bromides. The effect, in my experience, is very happy indeed; and, I may say, in accord with Dr. Lichty's experience, that I make it a habit to use, in all these cases, the cold spinal douche.

## THE GASTRIC DISTURBANCES OF OLD AGE

JULIUS FRIEDENWALD AND THEODORE H. MORRISON

*Baltimore, Maryland*

The gastric affections of old age are worthy of the most earnest consideration.

### ETIOLOGY

Of the various factors concerned in the etiology of these disorders the following are especially to be noted: (1) indiscretions in diet; (2) influence of diseases of other organs beyond the stomach itself; (3) infectious diseases; (4) focal infections; (5) degenerative changes; (6) arteriosclerosis; (7) syphilis; (8) toxemia, and (9) disturbances of the nervous system.

#### *1. Indiscretions in diet*

Habitual indiscretions in food and drink are often potent factors in the production of the gastric disturbances of old age. In this group the various forms of chronic gastritis and hyperchlorhydria are especially to be noted.

#### *2. Disturbances of the stomach due to diseases of other organs*

In a study of the gastric affections of old age one is particularly impressed with the fact that frequently the indigestion is secondary to diseases of some other and often distant organ. According to Fenwick, of every 100 cases of chronic dyspepsia in individuals over sixty-five years of age, 66 per cent are secondary to organic disease of some important organ, while but 34 per cent are due to progressive degenerative changes of the stomach and intestines. Among the diseases most frequently noted that may affect the gastric functions during old age are disturbances of the intestines, lungs, heart, liver, pancreas (diabetes) genito-urinary organs as well as the various forms of anemia and certain nervous affections.

### *3. Infectious diseases*

Beaumont first called attention to the fact that in febrile states there is diminished secretion of gastric juice with resultant lowered gastric digestion. It is therefore not uncommon to observe gastric symptoms such as anorexia, nausea and vomiting, accompanying such conditions as pneumonia, influenza and tuberculosis. Direct infections of the gastric mucosa with pneumococci and schizomycetes have been reported. It is important to note that the rôle played by infectious diseases in the production of gastric disturbances is not only important on account of the immediate effect, but that long after the infection has disappeared definite gastric manifestations may still remain as the result of the inroads made by the infection upon the stomach.

### *4. Focal infections*

It is a well established fact that focal infections may play an important rôle in the production of certain gastric disorders of old age. The primary forms may be localized in the teeth, tonsils, sinuses, gall-bladder, appendix, intestine, genito-urinary tract or other organs. It is generally conceded for instance that a certain proportion of gastric ulcerations are produced as a hematogenous infection with special strains of streptococci absorbed from certain foci of infection.

### *5. Degenerative changes*

The degenerative changes of old age are observed in the forms of weakness of the muscle fibres, together with atrophy of the mucous membrane resulting in atony and a diminution in the gastric secretion. Due to these alterations one frequently notes most distressing gastric symptoms often simulating malignant disease. The symptoms arise, however, as a rule, more gradually and frequently extend over a long period of time.

### *6. Arteriosclerosis*

Arteriosclerosis is a most important factor in the production of the gastric disturbances of old age. This process may involve the vessels of the stomach itself or the symptoms may be secondary to a general arteriosclerosis. As a result of the arteriosclerotic changes

a definite form of abdominal angina may occur associated with attacks of pain of a rather severe type. Degenerative changes in the gastric vessels may also result in gastric hemorrhages without the presence of any associated ulcerative lesion. A frequent cause of these hemorrhages is found in the miliary aneurysms due to arteriosclerosis of the small gastric arterioles.

### *7. Syphilis*

Syphilis of the stomach is not an infrequent affection in old age. In the largest proportion of instances, the cases have occurred in the tertiary stage of the disease, though symptoms of indigestion often accompany the secondary stage due to a toxic gastritis. Tertiary lues may, according to Fenwick appear in three forms as; (a) gummatous formations; (b) in the form of an endarteritis and, (c) as chronic inflammation of the mucosa.

### *8. Toxemia*

Toxemia associated with gastric disease is not uncommon in old age, and may be either of exogenous or endogenous origin. The exogenous forms are caused by the ingestion of poisons such as mineral acids, alkalies, phosphorus, arsenic and food infected with certain pathogenic microorganisms. The endogenous variety of gastric toxemia is far more common and occurs in such affections as dilatation of the stomach, carcinoma, chronic gastritis, intestinal obstruction, mucus colitis, intestinal stasis, dysentery, nephritis, anemia, pregnancy and in endocrine, liver and pancreatic dysfunction.

### *9. Disturbances of the nervous system*

The importance of the nervous system in its relation to the affections of the stomach in old age must always be held in mind. According to our experience 55 per cent of all gastric disturbances may be classified as functional, while but 45 per cent represent organic diseases. In order that a gastric neurosis may exist there must be present some predisposition, that is, a neurotic tendency in the individual. Heredity plays an important rôle. The neuroses rarely have their origin in old age. They begin more frequently in middle life and often continue on as individuals advance toward old age.

## THE GASTRIC SECRETION AND MOTILITY IN OLD AGE

From our observations as well as from those of Leifschutz and others, it is evident that the gastric secretion has a tendency to diminish in advancing years, and in a degree proportionate to the arteriosclerosis, and it is therefore unwise to attach too much importance to the absence of this secretion in individuals advanced in years, in the diagnosis of cancer of the stomach. This conclusion was recently again verified by one of us by means of fractional analyses of the gastric contents on 100 young patients and compared with the findings of individuals in old age affected with identical disturbances.

| YEARS    | NORMAL<br>ACIDITY<br>CASES | HYPER-<br>ACIDITY<br>CASES | SUB-<br>ACIDITY<br>CASES | AN-<br>ACIDITY<br>CASES | NORMAL<br>MOTILITY<br>CASES | HYPER-<br>MOTILITY<br>CASES | HYPO-<br>MOTILITY<br>CASES |
|----------|----------------------------|----------------------------|--------------------------|-------------------------|-----------------------------|-----------------------------|----------------------------|
| 20 to 40 | 45                         | 34                         | 12                       | 9                       | 55                          | 29                          | 16                         |
| 60 to 80 | 31                         | 24                         | 19                       | 16                      | 37                          | 14                          | 49                         |

From these observations it is evident that while in youth hyperacidity is more frequent than subacidity, that in old age hyperacidity is of less frequent occurrence and subacidity and anacidity are more common. On the other hand hypermotility is more frequent in youth and atony in old age.

TABLE 1

|  | NUMBER<br>OF CASES | PER CENT |
|--|--------------------|----------|
| Acute and chronic gastritis including achylia gastrica . | 28                 | 9.3      |
| Ulcer . . . . .  | 29                 | 9.6      |
| Cancer . . . . .   | 47                 | 15.6     |
| Dilatation . . . . .                                     | 23                 | 7.6      |
| Ptois . . . . .  | 41                 | 13.6     |
| Syphilis . . . . .                                       | 5                  | 1.6      |
| Secondary gastric affections . . . . .                   | 63                 | 21.0     |
| Nervous gastric affections . . . . .                     | 41                 | 13.6     |
| Visceral arteriosclerosis . . . . .                      | 17                 | 5.6      |
| Unclassified . . . . .                                   | 6                  | 2.0      |

THE INCIDENCE OF DISEASES OF THE STOMACH IN OLD AGE  
(REVEALED BY AN ANALYSIS OF 300 CASES)

In order to establish the incidence of the various gastric affections in old age an analysis of the records of 300 cases taken in succession was made and the results tabulated (table 1).

Arranged according to age and sex, the incidence of the gastric disorders of old age may be classified as in table 2.

According to the classification in table 2 the secondary gastric affections form the largest proportion of the gastric affections of old age, 21 per cent; next in number is cancer 15.6 per cent followed by the nervous gastric affections and ptosis 13.6 per cent; chronic gastritis and achylia gastrica 9.3 per cent; ulcer 9.6 per cent; dilatation 7.6 per cent and visceral arteriosclerosis 5.6 per cent.

#### ACUTE AND CHRONIC GASTRITIS; ACHYLIA GASTRICA

In our 300 patients of old age 29 or 9.3 per cent were affected with gastritis. Of these 6 presented the acute form while 22 were cases of chronic gastritis and achylia gastrica (atrophic gastritis). The acute

TABLE 2

| AGES         | DISEASES                    |        |       |        |        |        |            |        |        |        |          |        |                              |        |                            |        |                            |        |
|--------------|-----------------------------|--------|-------|--------|--------|--------|------------|--------|--------|--------|----------|--------|------------------------------|--------|----------------------------|--------|----------------------------|--------|
|              | Acute and chronic gastritis |        | Ulcer |        | Cancer |        | Dilatation |        | Ptosis |        | Syphilis |        | Secondary gastric affections |        | Nervous gastric affections |        | Visceral arterio-sclerosis |        |
|              | Male                        | Female | Male  | Female | Male   | Female | Male       | Female | Male   | Female | Male     | Female | Male                         | Female | Male                       | Female | Male                       | Female |
| <i>years</i> |                             |        |       |        |        |        |            |        |        |        |          |        |                              |        |                            |        |                            |        |
| 60 to 65     | 5                           | 1      | 9     | 4      | 18     | 10     | 4          | 3      | 4      | 11     | 3        | 1      | 13                           | 9      | 6                          | 9      | 3                          | 4      |
| 65 to 70     | 6                           | 5      | 7     | 3      | 7      | 4      | 9          | 2      | 6      | 8      | 1        | 0      | 14                           | 6      | 3                          | 13     | 3                          | 1      |
| 70 to 75     | 7                           | 4      | 4     | 2      | 4      | 4      | 2          | 3      | 2      | 10     | 0        | 0      | 18                           | 3      | 3                          | 7      | 5                          | 1      |
| Total . .    | 18                          | 10     | 20    | 9      | 29     | 18     | 15         | 8      | 12     | 29     | 4        | 1      | 45                           | 18     | 12                         | 29     | 11                         | 6      |

cases differed very little from those observed in younger individuals, and the condition was most common following dietary indiscretions and as a result of poor dentition. The same etiological factors were present in the chronic cases, and in addition abuse of alcohol and tobacco, neglect in the care of the mouth, and focal infection particularly of the teeth and tonsils seemed to play a part in the production of this affection. In this group of achylia only those forms were included in which a definite chronic atrophic glandular gastritis existed. Achylia of this type occur in pernicious anemia, carcinoma of the stomach, arteriosclerosis and at times in cholelithiasis and pancreatic disease.

## ULCER

Ulcer of the stomach and duodenum is not infrequently observed in old age and occurred in our series of 300 cases in 29 instances, that is, 9.6 per cent. In elderly individuals this condition occurred twice as frequently in males as in females. The diagnosis of ulcer in old age is not usually difficult. The characteristic symptoms such as pain, hematemesis, melena, positive X-ray findings and the presence of occult blood in the stools are noted usually in about the same proportion as in younger individuals. There is, however, a tendency to a far lower acidity in the gastric secretion than is noted in the young, and on this account the differential diagnosis in old age between ulcer and carcinoma may become extremely difficult. In most instances, however, a history of previous attacks of a similar type is obtained which usually aids in pointing toward the correct diagnosis. The treatment of ulcer in elderly patients differs but little from that in the young, except that in the former due to the state of subnutrition from the ulcer and the infirmities incident to old age, too great starvation and absolute rest in bed is contraindicated. For this reason a modified Lenhartz diet and only a partial rest cure is to be recommended. Since these patients, as a rule, are poor operative risks, surgical intervention is only indicated when complications arise, when the ulceration shows no tendency to heal under the usual treatment, and when the possibility of malignant transition is strongly suspected.

## CANCER

Cancer of the stomach occurred in 47 instances or 15.6 per cent in this series, 29 being males and 18 females. This lesion is more common in middle life than in old age for in 1000 cases studied by one of us 65 per cent was noted in middle life and 30 per cent in old age. Since there is a tendency to a diminution in the gastric acidity in old age, it is unwise to attach too much importance to this finding alone in the diagnosis of carcinoma of the stomach in elderly individuals. The presence of lactic acid, Oppler Boas bacilli, together with a positive Wolff Junghan's test for soluble albumin and a characteristic high blood sugar tolerance curve renders the diagnosis of carcinoma more certain in obscure cases. The roentgen ray may in addition give valuable information in arriving at a correct conclusion. It is

important to remember that in old age due to arteriosclerosis, retrogressive changes and impaired metabolism are not uncommonly present giving rise to a loss of strength and other symptoms simulating cancer. Further difficulties often arise by the occurrence of gastric cancer at this period of life in individuals affected with some chronic affection such as diabetes, nephritis or cardiac disease on account of which there is often marked emaciation, loss of strength and indigestion. The presence of a carcinoma may therefore be entirely overlooked in such individuals.

#### DILATATION OF THE STOMACH

In the study of our cases of dilatation of the stomach of old age, we have only included those cases in which a motor insufficiency of the second degree was noted, that is, where there were evidences of definite retention. This condition occurred in 23 instances or in 7.6 per cent in this series. Of these 6 were due to ulcer and 17 to cancer. The typical signs and symptoms were present in these cases, and in addition the diagnosis was corroborated by X-ray examinations. The long history of ulcer, absence of a palpable tumor and cachexia, together with the presence of free HCl and sarcinae in the gastric contents distinguishes the benign cases from those due to cancer. In the treatment of dilatation even in old age, early surgical intervention is usually indicated.

#### GASTROPTOSIS

Gastroptosis is extremely common in old age and is ordinarily associated with a similar condition of the intestine and frequently of other abdominal organs. It is also often complicated with other abdominal affections at times exerting evident influence upon the symptomatology of such disturbances. In our series this affection occurred in 41 instances, that is, 13.6 per cent; and only those cases were selected in which the symptoms manifested were almost entirely due to this condition alone. In many individuals of old age as in the young affected with this condition no annoying symptoms were observed. In the majority of cases definite manifestations were present which differed in no way from those observed in youth and middle life.

## SYPHILIS

Gastric syphilis is a rather rare affection, though it appears most frequently in middle life and old age. In this series it occurred in 5 instances, that is 1.6 per cent. It is much more common in males than in females. Clinically, syphilis of the stomach may be classified, according to Einhorn into three groups as follows: (1) ulceration of the stomach; (2) luetic tumor, and (3) syphilitic stenosis of the pylorus. These affections occur most commonly in the tertiary stage of syphilis, ulceration being the most frequent. The latter results either from the degeneration of a gumma or from the destruction of a localized area of the stomach produced by an endarteritis. The symptoms are similar to those of gastric ulcer, though relief is not afforded by food and alkalies. In those cases associated with a tumor and pyloric stenosis the symptoms are suggestive of cancer, but the Wassermann test will usually distinguish between the two conditions. The gastric analysis usually presents a true achylia even in instances of luetic ulceration. The X-ray findings also often strongly suggest carcinoma..

## SECONDARY GASTRIC AFFECTIONS

Disturbance of the functions of the stomach due to diseases of other organs occur more frequently in old age than in youth, and comprises the largest proportion of the cases in this series. In the study of our 300 cases of the gastric affections of old age, the secondary disturbances number 63 cases (21 per cent), occurring more frequently in males than in females. The secondary symptoms noted, which consisted largely of flatulency, fullness, constipation, belching, nausea, etc., are due to either a disturbance of the circulation, nervous system, or to a toxemia. The diseases in which disturbances of the gastric functions are particularly noted due to old age are tabulated as follows in our 63 cases.

|  | <i>Number of cases</i> |
|--|------------------------|
| 1. Acute febrile diseases.....                 | 2                      |
| 2. Diseases of the intestines.....             | 6                      |
| 3. Diseases of the liver and gall-bladder..... | 10                     |
| 4. Diseases of the pancreas.....               | 2                      |
| 5. Diseases of the kidneys.....                | 14                     |
| 6. Diseases of the heart and blood system..... | 16                     |
| 7. Diseases of metabolism.....                 | 2                      |
| 8. Diseases of the lungs.....                  | 7                      |
| 9. Diseases of the nervous system.....         | 4                      |

## NERVOUS GASTRIC AFFECTION

The gastric nervous disorders rarely begin in old age, they occur more frequently in youth and middle life. In this series, 41 cases were noted as nervous affections (13.6 per cent). The symptoms of the gastric neuroses in old age are not unlike those observed in younger individuals. The symptoms of a general neuroses are usually present, that is, irritability, lassitude, insomnia and depression. The subjective symptoms are changeable and capricious exhibiting protean changes in rapid succession. The digestive complaint is usually independent of the quantity and quality of food ingested, and frequently bears no relation whatever to meals. Frequent and sudden changes take place in the secretory and motor functions of the stomach or in both, so that a superacidity may quickly give way to a subacidity and a motor insufficiency to a hypermotility. In order to establish the nervous character of the gastric disorder, organic disease must be excluded. While any of the gastric neuroses may occur in old age, only the following are most frequent: cardiospasm, often secondary to gall-bladder disease; pylorospasm, often due to chronic appendicitis and at times simulating carcinoma; aerophagia, pneumatosis and bulimia.

## VISCERAL ARTERIOSCLEROSIS

Of our 300 cases of gastric disorder of old age, arteriosclerosis appeared in 17 instances (5.6 per cent). It occurred almost twice as frequently in males as in females. As an etiological factor concerned in the production of this condition is the natural wear and tear of life as is manifested in the changes in the blood vessels with advancing years. These changes are usually noted in old age; but this condition sometimes develops in those prematurely old, that is, even in middle life. There can be but little question, that the early onset of this affection is frequently due to such factors as over-work, mental over-strain, over-indulgence in food and drink, syphilis and other infectious diseases, and toxic factors such as gout, lead poisoning, alcohol and intestinal toxemias. Heredity plays an important rôle often, as one frequently observes early arteriosclerosis in members of the same family.

The main clinical gastric manifestations recognized in old age as the result of general arteriosclerosis and of arteriosclerosis of the abdominal arteries may be divided into three groups: (1) the dyspepsia due to general arteriosclerosis; (2) abdominal angina, and (3) gastric ulcer with or without hemorrhages.

### *1. The dyspepsia due to general arteriosclerosis*

The symptoms usually noted in this group take their onset insidiously, become more and more constant, the nocturnal attacks being particularly intense. These are manifested in the form of flatulency, fullness, distention, nausea, eructations, dizziness, palpitation and dyspnea. These symptoms may exist with a varying degree of intensity for years, and may even disappear for a short period of time to return with great severity. Dyspnea, flatulency and epigastric distress may appear following the slightest exertion, and without any relation to the ingestion food. Intestinal flatulency and distress now manifest themselves and the patient loses weight and strength, the symptoms progress and the patient may die either from an inter-current cerebral, cardiac or renal complication or from exhaustion.

### *2. Abdominal angina*

Abdominal angina occurs frequently as the result of an abdominal endarteritis. The pain which is similar to that of angina pectoris, is usually paroxysmal in character, is often increased by exertion or excitement, and is accompanied by tachycardia and hypertension. The pain which is caused by arteriosclerotic changes in the abdominal aorta may be manifested in the epigastrium, thorax or lower abdomen and is often transmitted to the dorsal and lumbar regions of the spine, and disappears under the influence of rest and the administration of the nitrites. When the gastric branch is involved in this condition, epigastric pain often of a very severe type is induced by a full meal, which is not entirely relieved until the stomach has fully emptied itself; light meals, produce but moderate discomfort.

### *3. Gastric ulcer with or without hemorrhage*

In old age, gastric ulceration is often due to a thrombo-angeitis of a branch of the gastric artery which causes marked ischemia of certain areas of the gastric wall thereby favoring the formation of this affection. Hemorrhages are often caused by the rupture of miliary aneurysms in the small gastric arterioles. The symptoms are similar to those observed in simple ulcerations. When in individuals in advanced years with evident manifestations of arteriosclerosis the usual signs of ulcerations occur, one should always suspect the presence of this affection.

In the study of the gastric disturbances of old age, the importance of arteriosclerosis should always be borne in mind.

In a patient of advanced years complaining of flatulency, distention and with epigastric pain and dyspnea which are relieved by eructations, especially when those symptoms are aggravated at night, a careful examination into the cardiovascular system should be made. Not infrequently there will be revealed marked hypertension, with an enlarged heart, with an accentuated second aortic sound or a murmur over the aorta, pulsation in the episternal notch, discomfort on pressure along the abdominal aorta, together with an increase in the urine which is of a low specific gravity, and contains albumin and casts. These findings may point directly to the arteriosclerotic changes as causative factors in the production of the gastric affection.

#### CONCLUSIONS

From our study of the 300 cases of the various gastric affections of old age the following conclusions becomes evident:

1. These affections differ in many respects from those occurring in younger individuals.

2. These variations are largely the result of infectious processes, degenerative changes and arteriosclerosis and are manifested frequently in changes in gastric secretion and motility together with other more or less characteristic symptoms.

3. In the study of the gastric disorders of old age these variations should be clearly borne in mind in drawing conclusions concerning the diagnosis and treatment of these affections.

#### DISCUSSION

DR. JACOB KAUFMAN, New York City: I should like to ask whether the diagnosis of syphilis was based on a positive Wassermann. I would say that I should doubt whether that is of sufficient importance to make a diagnosis on. I think that syphilis should be diagnosed on its characteristic features backed up by X-ray examination. I do not think that a Wassermann alone should be considered sufficient, because we get a positive Wassermann in cases in which, on postmortem examination, there are no evidences of syphilis.

Regarding arteriosclerosis in connection with angina abdominalis, I should like to mention a case reported by my former colleague, Dr. Schwyzer. At one time we palpated a tumor in the umbilical region, which disappeared after treatment given somewhere else. Several years afterwards, this patient used to suffer from attacks of extreme dilatation of the stomach by gas. The stomach was enormously inflated. On examination during the intervals, there was never anything irregular to be found. The gastric secretion and motility seemed to be normal. The attacks

were sudden, with gastric distention of an extreme degree. He had been examined by almost every man in New York, and no one could make a diagnosis. He died in such an attack. On postmortem, it was found that the former tumor had disappeared; and it was discovered that it was a case of syphilis. The tumor had disappeared under antisypilitic treatment, which had been given by the family physician. That probably led to the disappearance of a gumma, but there was still seen a typical scar in the omentum. The only other change found was from well developed arteriosclerosis of the gastric artery. That artery, and that one only, had its lumen almost occluded. The gastric symptoms were due to angina abdominalis. The artery would sometimes apparently close completely, and thus cause the stomach disturbance. Other things were found that proved that the man had syphilis, and a history of syphilis. The effect of the treatment on the tumor also was an evidence of this.

DR. GEORGE B. EUSTERMAN, Rochester, Minn.: I think that we are indebted to Dr. Friedenwald and his colleague for this instructive paper. The dyspepsia of senescence may be of different form from that of youth. I should like to ask what was meant by the statement that in dilatation surgery is necessary. I think that in a primary dilatation that is not secondary to pyloric obstruction, unless there is a retention of the barium (?) above ordinary limits, we should be careful about advising surgery. The diagnosis of gastric syphilis is absolutely as mentioned in the paper. We have had an experience to date with 60 cases. I am not egotistic about the amount of material we have; for I realize that a small number of cases studied thoroughly is as important as a large number studied superficially; but these were studied very intensively. Syphilis of the stomach is not uncommon. You must have a suspicious mind to that lesion, or you will overlook it. It is important that the internist recognize it as a clinical entity. I have only 1 case of proved syphilis of the stomach. Gumma practically does not exist. The syphilitic tumors that you feel are not in the stomach; they are in the omentum, the liver or the mesenteric glands. Occasionally circumscribed hypertrophy syphilis, inflammatory syphilis of the (colloid?) thyroid, may be found. Already I have had cases of ulcer associated with positive Wassermann and cases of cancer associated with this and other evidence of syphilis; but we have been able to establish criteria, which told us that even in the presence of a positive Wassermann, the lesion was not syphilis. What are those criteria? In the first place, the patient is younger than the cancer-bearing age. Eighty per cent of them are achylic; whereas in the others, only 40 per cent are achylic. When you have only obstruction in the pylorus, the filling defect is almost characteristic of syphilis. I have been able to tell most without any knowledge of the serology or history of the patient—in 99 cases out of 100—that the case is syphilis, from the X-ray aspect. Finally, these patients are even younger than the ulcer-bearing age. They have marked deformity of the stomach on X-ray examination; no achylia, as a rule; no obstruction; and the extent of their gastric pathology is out of proportion to the appearance of the patient. If it is a cancer, he is a sick-looking man.

DR. CHARLES G. STOCKTON, Buffalo, N. Y.: I do not know that I understand the paper clearly, as I did not hear the part dealing with syphilis; but it is a factor, I believe, as the walls of the stomach are seriously involved in the gumma. Prac-

tically all of us have had cases of marked rhinitis (?) in gastric conditions dependent upon syphilis. I have distinctly in mind 2 cases in which operation was ultimately necessary because of the extent of the rhinitis (?). One was operated on by Johnson, and came under my care for antisyphilitic treatment and local treatment of the stomach. The man is now enjoying reasonably good health. What I should like to have Dr. Eusterman explain a little more clearly is what he meant by saying that the walls of the stomach are not so much involved. How does he explain the filling defect in these cases, which are not without certain obstructive signs? I do not see how these can be explained by him as being in the liver or omentum.

DR. EUSTERMAN, (Speaking for the second time): I did not wish to give the impression that in syphilis of the stomach we do not have marked involvement of the gastric wall. No disease so extensively involves the stomach as syphilis. It was linitis plastica to which I referred. We do not know what that is, but it is probably a form of cancer. In cancer, it is more likely to encroach on the lumen and produce obstruction; but here it is a stiff tube, without retention or constriction. Surgery in these cases is almost impossible, because of the extensive thickening of the wall. We cannot do anything. It is a good thing that they do not obstruct; but in inherited syphilis they have the most extensive involvement, though there is no obstruction. Once in a while, you get an hour-glass stricture, but no obstruction. So I think that Dr. Stockton misunderstood me.

Regarding palpable masses, these are rare in the stomach except in thin patients, and then what you feel is a moderate thickening of the gastric wall. We can realize that we have recognized them clearly; because in cancer of the stomach with a positive Wassermann is the most confusing thing in which to differentiate between cancer and syphilis—the most confusing in gastro-enterology.

DR. JULIUS FRIEDENWALD, Baltimore (closing): With regard to the dilatation cases, I would say that these were all cases with stasis, and not simply atonic dilatation. The reason that we advised operation was because there was nothing else that we felt could possibly help such cases.

In regard to syphilis of the stomach, I agree in a large measure with Dr. Eusterman. In the first place achylia was always present. There was an absence of obstruction, and the X-ray signs were characteristic; and all these signs were present in those cases that we had here. In syphilis of the stomach, we do usually find this condition in young individuals; although we do occasionally find it in older ones. In some cases that I had under my care, there was definite obstruction; and pyloroplasty relieved the patient, and the cure was completed with antisyphilitic treatment. After all, the real criterion of the condition is the treatment, and the fact that antisyphilitic treatment cures these cases that we consider syphilis is rather definite proof that we have this disease to deal with.

## THE DIAGNOSIS OF INTESTINAL OBSTRUCTION

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Until an antidote is discovered for the toxemia of acute intestinal obstruction this lesion will remain the most fatal of all acute abdominal crises. The mortality has not been greatly diminished by aseptic surgery and whatever improvement may be noticed in the later statistics can be credited to the performance of an operation in an earlier stage of the condition, rather than to improvement in technical procedure. Recognition of the condition in this earlier stage therefore, becomes the most important factor in the cure of the patients by prompt operation.

Acute intestinal obstruction is the sudden arrest of the passage of the contents down the intestinal canal. Any one of many causes may be responsible for its development. The position and character of the obstructing force are important factors in determining the severity of the symptoms and the time of the fatal result. It is generally agreed that the higher the obstruction, the more severe the symptoms and the more rapidly fatal the disease. Strangulation, or other interference with the circulation increases the severity of the condition, at the same time adding to the probability of the fatal result. The more serious symptoms appear to be the result of the absorption of toxins from the obstructed bowel. The toxemia and the efforts of the gastrointestinal organs to overcome the obstruction produce a series of symptoms which are grouped together under the term ileus.

Ileus, therefore, may be the result of many conditions all of which obstruct the flow of the intestinal contents. Reflex and toxic paralyses, spastic contracture, as well as mechanical obstructions, either from within or without the bowel may be the underlying cause. The mechanical form always requires operation, the paralytic rarely; and the spastic type, while occasionally operated on, probably represents those cases in which spontaneous relief occurs.

The greater intensity of the toxemia in the presence of vascular disturbance is in part due to the resultant change in the intestinal wall. The peculiarities of the mesenteric circulation, especially the re-

sults of occlusion or patency of the parallel artery of the small intestine, play a large part in determining the effects of the obstruction (1).

It is difficult to imagine an obstruction of long duration from any cause in which the intrinsic blood supply has not been affected by the distension and distortion of the bowel wall. Such circulatory change may account for the sudden onset of acute symptoms in cases of long standing partial obstruction.

Whenever the underlying cause of an obstruction is of such a character that it can be recognized and removed early in the disease, the severity of the toxic symptoms is diminished, or their development is prevented. In strangulated hernia, where a painful external swelling calls direct attention to the obstruction, operation is performed in most cases before toxemia develops. The irreducible external swelling focuses attention on the obstruction and brings the patient to operation without delay. Although certain other conditions may be responsible for the protrusion these are quickly excluded, or recognized as lesions which require operation for their cure.

Similar reasoning should bring the internal obstructions to early operation. In spite of the fact that there are no signs so distinct as the external hernial swelling, the symptoms of intestinal obstruction are well defined and with proper interpretation it is often possible to make a diagnosis before the onset of grave toxemia.

It is convenient to divide the symptomatology and course of acute intestinal obstruction into three stages.<sup>1</sup>

1. The stage of onset, when the symptoms are due to the arrest of the intestinal current.

2. The stage of compensation, when the gastrointestinal organs attempt to overcome the obstruction or its results.

3. The stage of sequellae or complications, when the obstruction has caused secondary destructive changes in the bowel, or in the body as an entirety.

The higher and more complete the obstruction the less clearly defined will be the symptoms of the different stages. In most cases there will be a merging of the symptoms of the various stages. Vascular injury always intensifies the course of the disease. Gangrene of the bowel may either complicate the compensatory effort, or be present with the toxemia.

<sup>1</sup>These stages do not represent any definite period of time. In twenty-four hours a patient may pass through all three and die of toxemia.

The characteristic symptoms of onset are pain, constipation or diarrhea, and vomiting. The pain is paroxysmal, with free intervals, or it may be continuous between the exacerbations. This paroxysmal pain usually described as cramps, is the most constant initial symptom and becomes increasingly severe during the first hour. The pain of obstruction is not relieved by defecation or by vomiting. Opium brings only temporary ease. Constipation may be absolute, or there may be an initial bowel movement preceding the onset of pain. The constipation resists well given enemas, or an effectual enema does not give relief from the pain. Tenesmus, or diarrhea and pain with blood and mucus in the stools occur in certain cases of strangulation with intussusception and with intestinal tumors.

Vomiting may be the initial symptom, followed by pain and constipation, but the usual sequence is pain, vomiting and constipation. The vomited material may be gastric or duodenal contents. As a rule, however, the material is progressively gastric, bilious, and then intestinal. At the onset it is gastric, occurring without regard to the ingestion of food. Lavage does not relieve the symptoms. The initial vomiting is reflex, and may be replaced by hiccough.

The blood pressure practically always falls as the symptoms develop. Occasionally there is a rise in the pressure during this initial stage. A falling pressure indicates the onset of intoxication.

The symptoms of the second stage are local and general. The pain becomes more intense unless gangrene develops, in which event it may be diminished. The vomitus of the second stage consists of material flowing back into the stomach from the obstructed intestine. The quantity vomited may be extremely large. There is a great increase in the amount of fluid secreted by the obstructed intestine. The fluid is thin, acrid, disagreeable in taste and odor, and is irritating to the mouth, lips and chin. The color seems to darken with the duration of the obstruction, passing from yellow through green to dark brown.

Visible peristalsis or visible and palpable stiffened intestinal coils are the most characteristic symptoms of the second stage. The stiffened coil is a distended area in which the contractions have reached the tetanic stage, and in which there has occurred an infiltration of the wall with an accumulation of fluid and gases in the interior of the bowel. Visible peristalsis is due to an extraordinary activity of the bowel produced at this time. The movements are vigorous and associated with the presence of the visible stiffened coil. When much small intestine is involved in the process, the spastic distended coils

may show as a series of parallel ridges, the ladder pattern. The contractions are accompanied by pain unless the toxemia has become grave.

The early distention is regional and usually asymmetric. Later there will be general distention. Tympanites accompanies distention. The lower the obstruction the greater the distention and tympanites. Local tenderness may indicate the soreness of spastic muscle, or may be from a local peritonitis at the point of obstruction.

The blood pressure falls during the second stage.

In the third stage the symptoms are those of toxemia, gangrene, peritonitis and alteration of the function of the liver and kidneys.

The toxin of intestinal obstruction is a virulent poison, although there appear to be individual variations in resistance to it.

The rate of the pulse and respiration is increased, the blood pressure falls; there is great prostration and cyanosis with or without clammy skin. The mental condition varies from noisy delirium to unconsciousness. Often there is a subjective sense of well being, contrasting greatly with such objective signs as regurgitation of stercoraceous material, and distended abdomen in which violent peristaltic action is visible.

The development of gangrene is accompanied by collapse, and often sudden and severe increase of pain, although later the pain is diminished. As a result of leakage peritonitis ensues and with its spread comes movable dullness in the flanks and obliteration of liver dullness.

Peritonitis is the result of invasion by the intestinal bacteria either through gross rupture of gangrenous bowel, or by penetration of the bacteria through an area of intestine whose wall has been altered by distention or constriction. The absorbed toxin probably reaches the circulation through the lymphatics. The liver seems to be involved in the defense of the body, but the changes produced in this way are not yet explained. The kidneys probably make an attempt to excrete the toxin from the blood, and in this way certain changes in their function are brought about. The intestine itself is involved in this process of excretion, which therefore brings about a vicious circle.

Laboratory examinations are not of great value in diagnosis, although the following estimations may prove helpful.

*The blood count.* Usually there is a marked leucocytosis. In a series of 62 cases the average was 16,000; in but 2 was it below 5000, and in 7 others below 10,000. The highest was 46,000 in a case of volvulus of the sigmoid (2).

*The non-protein nitrogen in the blood.* In obstruction the quantity of this nitrogen in the blood is increased. Certain variations have been noted by experimenters which indicate that a series of observations made every four or six hours will give better information than a single estimation. If the successive examinations show an increase operation is indicated, while in the presence of a falling or stationary quantity delay seems justifiable (3), (4).

Unfortunately many of the lesions in which a differentiation from obstruction is difficult give similar changes in the blood nitrogen, or make a diagnosis so urgently necessary that delay for repeated examinations cannot be afforded (5), (6).

*The urine.* Albumin and casts are frequently present from the onset of symptoms and are found in all cases of severe toxemia. The casts disappear from the urine after the obstruction has been relieved (2). Indol and skatol derivatives are present in the urine in obstruction, but they are also present in so many other conditions that their estimation is of no value in diagnosis.

The foregoing are the general symptoms of intestinal obstruction. No attempt has been made to give signs which will separate the variations in the condition which depend on differences in etiological factors. Such signs exist and usually may be interpreted with ease, but no time should be lost in searching for such differential points. The diagnosis of acute intestinal obstruction as an entity is sufficient indication for operation.

The differential diagnosis between intestinal obstruction and other lesions having similar symptoms becomes most important when we are called on to separate the conditions which require operation for their cure from those in which operation is not necessary, or in which it may be even harmful. In the former group of cases it may be impossible to make an accurate diagnosis beyond the recognition of the crisis, which demands immediate intervention. The exact nature of the trouble in such cases must be decided after the abdomen is open. Delay for academic accuracy of diagnosis wastes the opportunity for curing the patient before the development of complications which make the use of disagreeable expedients necessary.

*Abdominal symptoms of thoracic disease.* The onset of pneumonia, of diaphragmatic pleurisy, of pulmonary infection, of pericarditis, as well as the acute exacerbations of cardio-vascular disease may be ushered in by marked abdominal symptoms, which may closely simulate those of the first stage of intestinal obstruction, or be accompanied

by distention and other signs of the toxic stage (7). Throat infections in children may simulate abdominal disease (8). X-ray examination of the chest will often show the lung lesion and will aid in recognizing the cardio-vascular ones when physical signs are indistinct. Levine (9) notes in the confusing cardiac lesions a small thready pulse with slight increase in rate and a regular rhythm, but quite distant heart sounds. The development of a slow pulse and fainting attacks points directly to a cardiac condition. If the patient is over forty with a history of dyspnea and there are any physical signs of cardiac enlargement, the probability of a thoracic rather than an abdominal cause for the symptoms becomes greater. When the apparatus is available, the electrocardiogram may add to the certainty of the heart lesion.

If the area of pain and tenderness be carefully mapped out, and the patient then be directed to hold his breath, the pain and tenderness will be absent as long as the diaphragm remains quiet (10). In the absence of other signs of thoracic disease, abdominal pain associated with movements of the alae nasi during respiration indicates a thoracic and not an abdominal lesion. The anxiety in the thoracic lesion is lethargic and resigned, not terror stricken and active as in the abdominal conditions (11). If the possibility of thoracic disease as the cause of the symptoms is kept in mind, a mistake in their interpretation is unusual. During an epidemic such mistakes are rare (12).

The crises of tabes are often associated with distention of the abdomen. The characteristic changes in the pupil and in the other reflexes make the recognition of the tabes definite.

The visceral crises of angioneurotic edema may simulate an acute obstruction. The presence of blood in the stools may lead to the suspicion of intussusception or tumor. The history of previous attacks, or the association with symptoms of hives, of purpura, or other superficial manifestations of the condition will make the diagnosis. Blood in the urine is suggestive of the angioneurotic lesion rather than obstruction (13). The non-proteid nitrogen of the blood is not increased in tabes or in angioneurotic edema.

*Acute dilatation of the stomach.* In this condition the pain is continuous and referred to the chest wall. The distention is epigastric. There is no vomiting, but rather a regurgitation of discolored fluid. Lavage gives immediate relief from the symptoms.

*Adrenal disease.* Destruction of the adrenals by disease and experimental removal of these bodies both give rise to certain symptoms resembling those of the toxic stage of obstruction. The action of the

toxin of obstruction is almost identical with that of acetylcholin, which substance acts as a physiological antagonist to the suprarenal secretion. The development of analogous toxic symptoms after the destruction of the adrenal bodies points to some antagonism between the substances and indicates the value of epinephrin in the treatment of patients suffering from obstruction with toxemia. The diagnosis between the conditions can only be made by identifying the preëxisting adrenal disease.

*Spastic ileus.* Spasmodic contracture of a segment of the bowel occurs in lead poisoning, in certain tabetic crises and as the result of a number of reflex irritations. The obstruction may become so complete as to lead to toxemia and death. As a rule the symptoms are intermittent, but they may be continuous and rapidly progressive. The condition is often observed after operations in or near the peritoneal cavity, for example, after lumbar nephrectomy. Apparently a spastic obstruction may be the beginning of an intussusception, the contracted segment being thrust into the lower loop (14). The differentiation between spasmodic and mechanical obstruction can not be made from clinical investigations and therefore in all cases where the symptoms are urgent, operation should be performed and the diagnosis made after the abdomen has been opened. The spasm usually relaxes spontaneously, or as the coils are handled. This fact may account for some of the cases diagnosed as obstructive in which laparotomy has failed to reveal any lesion.

*Mesenteric vascular occlusion.* The occlusion may be arterial or venous, embolic or thrombotic. A combination of injury and infection is the usual cause. The symptoms are very like those of acute obstruction. At the onset the vascular lesion is usually associated with greater shock and there is likely to be a passage of a considerable quantity of blood and fecal material. The distention is general and is seldom as great as in obstruction. Some cases diagnosed mesenteric thrombosis or embolism have recovered without operation, but as a rule it is better to open the abdomen and deal with the condition surgically.

*Acute pancreatitis.* The history of long standing gall-bladder disease in an obese individual, who has an acute abdominal crisis, especially if the attack is associated with cyanosis and great collapse, will lead to the suspicion of pancreatitis, but the diagnosis is never certain until the fat necrosis is found after the abdomen has been opened. Therefore no time should be lost in attempting a distinction between

this lesion and an acute obstruction. If, as Ellis (15) indicates, there is a close relation between the toxin of acute obstruction and that of pancreatitis, the differentiation between the lesions approaches the impossible.

*Ruptured abdominal viscus.* After the original collapse, the symptoms of ruptured viscus are those of spreading peritonitis rather than of obstruction. The continuous pain and the board like rigidity of the abdomen are the best signs for differentiation. Similar symptoms are associated with rupture of an ectopic pregnancy. In this lesion the menstrual history usually helps. The signs are more marked in the lower abdomen, and a pelvic examination may make the diagnosis certain. Bluish discoloration of the umbilicus when present indicates ectopic pregnancy (16).

Torsion of a pedicled tumor may give symptoms resembling those of obstruction. In the early stage the presence of the tumor will indicate the character of the lesion. In the late stage, a reflex paralytic ileus may complicate the picture. While this condition makes accurate diagnosis doubtful, it gives a clear indication for the necessary treatment, namely, operation.

The onset of appendicitis, of cholecystitis and certain types of kidney colic may give symptoms very like those of acute obstruction. With the inflammatory lesions there is usually fever, and there is less shock than in obstruction. The character of the pain is continuous and there is local tenderness and much spasm from the onset of the attack. Appendicitis in older persons is likely to begin with obstructive symptoms (17).

Kidney colic is associated with blood in the urine, either grossly or microscopically. There are usually other symptoms referred to the urinary tract, as for example, vesical tenesmus.

The differentiation between peritonitis and intestinal obstruction is usually difficult and often impossible. Laboratory examinations do not help and the clinical signs are likely to be confusing. A local peritonitis by infiltration of the bowel wall can set up an obstruction. The fulminating type of peritonitis causes a paralytic distention of the intestine with stoppage of its contents. A tightly strangulated bowel will allow the passage of infectious material through its damaged wall and thus set up a local peritonitis.

At the onset the continuous pain of peritonitis contrasts with the paroxysms of obstruction. Muscle spasm and rigidity of the abdominal wall are prominent symptoms in peritonitis and are absent in

obstruction. Fever is more likely to be present in peritonitis, and peristalsis is lessened. The development of distention in a quiet abdomen indicates peritonitis, and makes a contrast to the noisy abdomen of obstruction with its visible peristalsis and palpable distended coils. The distinction between peritonitis and obstruction is most important in post-operative cases. Occasionally the nature of the primary operation gives a clue as to the more probable complication. It is possible that a rapidly increasing quantity of nonprotein nitrogen in the blood may be characteristic of obstruction rather than of peritonitis.

With these facts in mind therefore, how shall we proceed when called on to treat a patient who is suddenly seized with paroxysmal abdominal pain, nausea or vomiting, and disturbance of the bowel movements.

The onset of an acute thoracic lesion, or one of the cardiac upsets should be considered and decided. Unless the lesion above the diaphragm is definitely recognized, the attention should be focused on the abdomen. Lead colic, angio-neurotic oedema and tabes are recognized by their extra abdominal symptoms.

The physical examination will determine the presence or absence of masses, tender points, local distention, etc. A ballooned rectum suggests a low obstruction. An empty rectum, or one containing only a little feces, is found with obstruction of the small intestine. The presence of a large quantity of feces, especially if the material be hard, indicates a coprostasis rather than an obstruction. Severe symptoms however are rare in cases of fecal impaction. The obstructed loop may be felt through the rectum.

An enema should be given by a competent person, and the stomach emptied by lavage. If an effectual enema does not bring relief from the pain, the suspicion of a mechanical obstruction becomes very strong. Similarly gastric lavage which does not bring relief points to an obstruction. If the pain continues, both enema and lavage should be repeated after an hour. If the second enema is retained, or escapes unaltered and with slight force, the presence of an obstruction becomes certain. If the second lavage brings away duodenal contents, the diagnosis is made even more certain.

As a rule these tests are sufficient to lead to a diagnosis. In 18 cases of post-operative intestinal obstruction an operation was performed after the diagnosis had been made on these symptoms. In every case a mechanical obstruction was found and relieved and all these patients recovered.

Should the effect of the enema and lavage fail to be convincing, corroborative evidence of obstruction may be furnished by a rise in the quantity of the non-protein nitrogen in the blood. In the doubtful cases repeated blood studies at four hour intervals are valuable. During this period of doubt the patient should be in the hospital, prepared for operation. The time may be employed in repeating the enema and lavage.

Cathartics are so dangerous that it has become a rule to prohibit their use in the presence of symptoms of obstruction.

Opium is banned, because it may mask symptoms.

In managing the patient in whom an obstruction is suspected, the judicious use of opium or a cathartic may be employed during this period of doubt. After the stomach has been emptied by lavage 2 or 3 ounces of castor oil is poured in through the tube. At the same time  $\frac{3}{4}$  of a grain of eserin salicylate, or a dose of pituitrin is given hypodermically. In the presence of mechanical obstruction this treatment intensifies the primary symptoms and hastens the development of the secondary ones to such a degree that the diagnosis becomes certain, and operation imperative.

On the other hand, relief of the symptoms ends the uncertainty.

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## DISCUSSION

DR. GEORGE B. EUSTERMAN, Rochester, Minn.: This is a very important subject to all of us, and the only thing I want to say is that I hope this excellent paper will be recorded, with all his notes, in the minutes of the Proceedings.

DR. JOHN C. HEMMETER, Baltimore: I failed to hear what Dr. McGlannan said about the leukocytes.

DR. MCGLANNAN: "C 5" showed leukocytes, 36,000. It was in a volvulus. I feel that we can not draw definite conclusions from the leukocytes.

DR. JAMES TAFT PILCHER, Brooklyn: There is a point that I think will be elucidated in the complete paper, although the doctor did not mention it; and that is, the most extraordinary phenomena of acute intestinal obstruction in cases of abdominal angioneurotic edema. I have had 2 cases. I wished to call attention to this, because it is possible that Dr. McGlannan may have neglected to mention it in his paper.

DR. ALBERT F. ANDRESSEN, New York City: I think that this subject is of greater importance to the general practitioner than are many others, because these cases usually go first to a general hospital. They come to us extremely late, when they present the typical symptoms that the textbooks describe as being the symptoms of intestinal obstruction. These are not, however, the ones that Dr. McGlannan has described. The textbooks stress fecal vomiting, abdominal distention and the Hippocratic facies. When the patient reaches that stage, he is, as a rule, beyond all hope of surgical interference. I think that our textbooks should be improved in that respect, and that Dr. McGlannan did well to leave the symptoms that they give out; because they are symptoms of dissolution, although they are the ones stressed in all textbooks.

Dr. McGlannan says, and I think it should be emphasized, that castor oil by mouth should be given in the hospital, and not outside, by the general practitioner. I think that most of the patients that we see have had their doses of castor oil and salts and enemas before reaching the hospital; so that the treatment would not be indicated further. But in all cases seen early, the effort should be made. I think that if it is not too late, we should use castor oil, enemas or olive oil to soften a possible impaction, and follow it by soap suds in an hour or two.

Another thing that does not add to the risk of waiting is to radiograph and fluoroscope the patient without giving a meal. Sometimes this will show clearly the point of obstruction as revealed by the distended bowel. I know of 1 case in which the surgeon was greatly aided in knowing where to make the incision in carcinoma of the descending colon by observing that there was gaseous distention right down to the point of obstruction, and none below it.

DR. A. McGLANNAN, Baltimore (closing): There is a good deal more written in the paper than I read; because when you start on the subject of intestinal obstruction, it is hard to know when to stop, and cover the subject reasonably well, without tiring the patience of the audience.

These late symptoms are distressing and easily recognized; but all the textbooks speak of them; and it does the patients no good, because they die. The X-ray would certainly be an aid in knowing just where to go in. Usually we go in on the right side, because most obstructions are in the neighborhood of the right lower quadrant; but it is not always so, and sometimes it is necessary to make a second incision.

Fecal impaction may be diagnosed by the finger in the rectum. It hardly ever gives severe symptoms. We are not worried about a patient with fecal impaction. He does not get very ill.

# INTESTINAL INFECTION WITH ENTAMOEBA HISTOLYTICA AS A FACTOR IN ARTHRITIS DEFORMANS

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In the California State Journal of Medicine, of February, 1922, Kofoid and Swezy present a brief report of a case of arthritis deformans, in which the protozoal organism, *entamoeba histolytica*, was found, in the bony structures of the hip joint, removed at operation, by Professor L. W. Ely, of Stanford University. No claim is made in the report, of a comprehensive study of this interesting finding, though the authors promise further research, and assurance is given, of the publication of section photomicrographs in a later communication. A plea is likewise put forth, by the authors, for a more general examination of specimens of feces in all cases of arthritis deformans, for the purpose of detecting the possible presence of an *Entamoeba histolytica* infestation of the intestinal tract. Since the publication of this article, I have had the opportunity of observing a case of advanced arthritis deformans, of the atrophic type, in a patient, in whom an active intestinal entamoebiasis has been demonstrated. It is needless to say, that the confirmation of a direct causal relationship, existing between an entamebic infection and the destructive lesions of arthritis deformans, would present possibilities of great importance in clinical medicine. The essential lesion of arthritis deformans, has been described by Ely, in previous communications as consisting of a primary necrosis, of the bone marrow in the region of the joint. Subsequent changes involve a hyperplasia of the bone and cartilagenous tissue around the necrosed area, which eventually degenerates, and wears away thus leaving an articular surface of dense, eburnated bone. This constitutes the type described by Goldwaite as hypertrophic or osteo arthritis. In the atrophic form, often spoken of as rheumatoid arthritis, inflammatory proliferation occurs principally in the joint capsule and especially in its synovial lining. The pathological process, presented by both types differs in many respects from the arthritides produced by such organisms as

the streptococcus, the tubercle bacillus or the *Treponema pallida*. Attempts have frequently been made in the past, to obtain cultures from the synovial fluid and from the necrotic bone or cartilage in cases of arthritis deformans, without success. The diseased areas in fact have been found consistently sterile though the supposition is that the original infectious bacterium has become attenuated or has been killed off entirely as a result of subsequent pathological changes. In the absence of a demonstrable organism, the etiology of the disease has always remained cloudy, though various hypotheses have been advanced, including remote foci of bacterial infection in teeth, gums, tonsils and other organs, indeterminate intestinal toxemias, as also the contributing effect of certain atmospheric changes. The discovery of the *Entamoeba histolytica*, in the region of the necrosed area of the bone in the case reported by Kofoed and Swezy brings forward a new conception of the etiology of the disease. Barrow was the first to suggest the possibility of this relationship, the idea occurring to him as a result of finding pathogenic entamoeba in the stools of one of Ely's cases.

It is interesting to note, however, that as far back as 1916, Moorhead discussed the occurrence of arthritis as a complication or sequel of amebic dysentery. This author reported in detail, 6 cases of progressive polyarthritis, in connection with chronic dysentery, in 4 of which typical forms of *entamoeba histolytica* were present in the feces. He likewise, suggested that in all cases of arthritis, a history of dysentery should be inquired into, and, if obtained, advised that a course of emetine therapy be instituted at once. Moorhead did not attempt to demonstrate the presence of the organisms, either in the synovial fluid, or in sections of the joint tissue, removed by operation. In the recent case, reported, by Kofoed and Swezy, this further and more definite proof of the direct invasion of the joint structures, by the entamoebae, was demonstrated. In this instance, a portion of the head of the femur was removed at operation, fixed in formalin solution, decalcified, and sections stained in iron hematoxylin. The entamoebae were found on the outer circumference of the areas of necrosis, in greatest numbers, around the peripheral capillaries. The stained organisms presented the typical nuclear structure of the *entamoeba histolytica*. In some instances, even the characteristic pseudopodia could be made out. The nuclear arrangement was distinct from that presented by other forms of parasitic entamoeba or of tissue cells, either normal or pathological. The organisms confirmed

in every respect, to the type previously described by Councilman and Lafleur, as present in the tissues surrounding entamoebic ulcerations of the intestinal tract. While the pathological evidence in this case, of a remote infection of the joint structures with *Entamoeba histolytica* would appear convincing, it must not be overlooked that, in the past, mention of a clinical relationship between the two conditions, has rarely been recorded. Garrod, for example, writing, in *The Allbut System of Medicine*, claims that while arthritis is undoubtedly frequently met with in bacillary dysentery, he was unable to find any definite evidence as to its occurrence in the amebic form. Such well known authorities on entamoebiasis as Brown, Manson, Strong or Rogers, fail to make mention of arthritis as a complication of the disease. On the other hand, numerous references to arthritic complication in bacillary dysentery is by no means infrequent. In 400 cases of arthritis, recently studied by Pembroke, based on experiences in the Army, dysentery was given as the causative factor in 33 or 8.25 per cent. The type of dysentery was not stated, though presumably it was of bacillary origin. This author is credited further, with the significant statement, that arthritis may follow infection with yeast and fungus growths, and also with the *Entamoeba buccalis* though the latter is not certain.

A further interesting problem in connection with the invasion of the bone marrow by *Entamoeba histolytica*, is involved in the route which the organism traverses. Infection with the *Entamoeba histolytica* occurs primarily in the intestinal tract. Invasion of the liver, which constitutes the most frequent form of secondary complication in entamebiasis follows the path of the portal vein. In some few instances the organism has been known to reach the brain, 35 cases in all this remote complication having been reported, in the literature to date. Invasion of the spleen is of even less frequency, and arises most probably from contact with lesions in the transverse and descending colon. Cases of pulmonary amebic abscess can always be traced to the liver. In the case of cerebral involvement, the entamoeba probably travel from a focus of infection in the liver through the vena cava into the pulmonary artery, finally reaching the brain through the arterial system. A similar mode of arterial distribution must occur in the event that the entamoeba find lodgment in the bone marrow. In the case reported by Kofoed and Swezy, it is interesting to note, that no mention is made of clinical complications other than the arthritis.

In the following case of chronic arthritis deformans, complicated by an intractable type of dysentery, the presence of the vegetative entamoebic histolytica was demonstrated in the feces.

T. S., male, aged twenty-seven, married, auto salesman by occupation, a native of Texas, was admitted to Touro Infirmary April 18, 1921. The past history of the patient had been uneventful. Outside of the usual diseases of childhood, his health had always remained good. During the World War, he served in the airplane service, in his native state of Texas, for a period of one and one-half years. During this time, he claims to have lived mostly in the open, and was exposed to all types of weather changes. To this, he attributes the onset of his troubles. The arthritis appeared in definite form, however, eight months previous to his admission to the Infirmary while the patient was sojourning in Cuba. The disease attacked the knee joints first, spreading rapidly to the ankles, toes, wrists, elbows, fingers, shoulders, hips. The course pursued was that of a subacute infection. The inflammatory process and temperature changes followed in the beginning only a mild course. The treatment at this time, consisted of thermal baths and salicylate medication, which brought little relief. Within two months, the patient became bedridden. In January, 1921, he came under observation in a local hospital, and at that time, all sources of possible focal infection were carefully sought for. The tonsils were subsequently removed, and autogenous vaccines prepared therefrom, 5 injections in all having been administered. The teeth and gums were likewise reviewed by a competent dentist, and a number of teeth with infected pulp canals were extracted. Slight improvement followed these procedures, but proved only temporary. He was admitted into Dr. Matas' service, approximately one year ago, and though he has received the benefit of most painstaking treatment, involving various well established methods, no improvement has been noted. His present status is one of extreme emaciation with a marked degree of anemia present. The weight registers 45 pounds under normal (165 pounds). Blood picture, red count 2,950,000. No special abnormalities have been noted in the urine. The joints present the characteristic appearance of the atrophic type of arthritis deformans. Marked deformity with ankylosis is present with diffuse infiltration of the entire joint tissues and degeneration and erosion of the cartilage. The skeletal muscles are markedly atrophied. The joints are tender to touch, and the patient experiences keen pain on the slightest movement. The dysenteric complication in the case began about four months following the onset of joint symptoms. Prior to that time, the bowel functioning had been normal. The number of daily dysenteric bowel evacuations have varied, but average about four in twenty-four hours. The character of the stool is distinctly dysenteric, being composed largely of blood, mucus and pus. Considerable tenesmus accompanies each evacuation, and in recent months a tendency to spontaneous evacuation has been shown. The *Entamoeba histolytica* in active form was discovered in the stools in February, 1922. A course of ipecac medication by mouth and of emetine hydrochloride hypodermatically was immediately instituted. Because of the patient's generally unsatisfactory condition, full dosage with these drugs was deemed inadvisable. The use of the specific medication, however, has removed all evidence of the protozoal infection in the feces at the present time. Nevertheless, in spite of this, the dysenteric stools have continued and large amounts of pus and blood are still present in the

stools. Proctoscopic examination has been persistently refused by the patient, though presumably an extensive ulceration of the large bowel exists. No change in the condition of the joints was noted following the administration of the ipecac and emetine. We have likewise been unable to obtain any of the synovial fluid or joint tissue for examination for the presence of the protozoal organisms.

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#### DISCUSSION

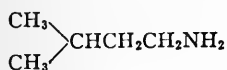
DR. JOHN C. HEMMETER, Baltimore: Last week, I sat between Drs. Welch and Warne at dinner. The Librarian of the Surgeon General, Dr. Noble, asked me to read a biography of Dr. Woodward, who wrote the "Medical History of the War of the Rebellion." It is, without doubt, the greatest account of intestinal diseases in the English language. He did not have any personal experience with *Enamoeba histolitica*, but he pictured it in photographs and gave an account of Lesch's experience with it. Dr. Simon will find in the History the number of cases of rheumatism and abscesses of the liver that occurred with dysentery. The book is also the completest history of dysentery. It does back to Herodotus and shows the relation of dysentery with infection in people who were entirely ignorant of the subject of infection.

# INTESTINAL TOXEMIA: ENTEROTOXINES ALLIED TO ADRENALIN IN COMPOSITION WHICH CAUSE RISE IN BLOOD PRESSURE AND INCREASED SYMPATHICOTONUS

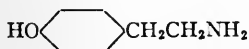
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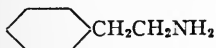
When one compares the chemical formula of certain products of intestinal putrefaction such as leucine, tyrosine and phenylalanine with the chemical formula of adrenalin one is struck with the resemblance of the stereochemic structure. In order to illustrate this I will arrange the formulae of the intestinal enterotoxines just mentioned with adrenalin for sake of comparison.



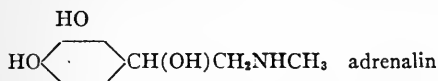
Isoamylamine



p-hydroxyphenylethylamine



phenylethylamine



Leucine would yield isoamylamine, tyrosine would give rise to p-hydroxyphenylethylamine and phenylalanine would yield phenylethylamine. There is a series of substances more or less allied to adrenalin which are produced during the putrefaction of meat and Dale and Barger have identified them as forming the most important of the active principles of ergot. All of these substances have a more or less vaso-constrictor effect to which, in fact, their pharmacologic activity is due. They produce constriction of blood vessels, contraction of involuntary muscular fiber. By virtue of this effect these substances can raise blood pressure and as this is one of the physiologic effects of stimulation of the sympathetic nerve fibers supplying the muscles in the walls of the arterioles, these substances

have been called "sympathicomimetic" which means mimicing the sympathetic nerve. Among the toxic-monamines which produce the effect of a sympathetic stimulation, especially rise of blood pressure is p-hydroxyphenylethylamine, this toxic-amine could be formerly obtained in Europe in the form of the phosphoric-acid salt and under the name of tyramin.

It is formed in the lower ileum and colon from tyrosin under the influence of anaerobic, facultative intestinal bacteria. In the liver this toxamine is transformed into p-hydroxyphenylacetic acid and in this form it is excreted in the urine and can be demonstrated there by a method to be given later.

The blood pressure raising effect of this monamine amounts to one-fifth to one-fourth that of adrenalin. When 30 to 50 mgm. of p-hydroxyphenylethylamine is injected into the circulation of a healthy human being, it causes a systolic rise of blood pressure that reaches its height in ten to fifteen minutes and disappears in forty-five minutes to one hour. There are individuals in which there is a latent period of a half an hour before the rise of blood pressure begins but they are very rare. In such persons, in which there already exists a hyper-tonicity of the sympathetic nervous system, the rise begins sooner and reaches a greater height. In all cases the rate of the heart beat becomes slower prior to the rise of blood pressure.

There are human beings who continually form this enterotoxine in their intestines and who develop various forms of diseases which on superficial observation apparently resemble widely different clinical conditions. For example, the ceaseless absorption of this monamine has in my experience produced such widely different symptomatology as that of paralysis agitans and Basedows disease and gout. Why this varying effect should occur in human beings cannot be at present determined. It very likely depends upon the greater or lesser degree of intestinal intoxication and also, upon the admixture with endocrine disturbances already present in the individual or developed upon some organ of lessened resistance. For instances, there are cases where focal necrosis has occurred in the liver, this being the first organ which the p-hydroxyphenylethylamine reaches after its absorption from the intestine. Prolonged constriction of the terminal branches of the hepatic artery will deprive the district of hepatic-lobules of oxygen which that arteriole supplied. Without oxygen the hepatic cells cannot live because the portal vein blood does not carry enough oxygen to maintain the life of the liver cell.

It is my opinion that there are types of focal necrosis, which may lead to hepatitis which are due to the prolonged absorption of this monamine from the intestine. That such a hepatic disorganization, if it extends over more than one lobe may bring on a cirrhosis of the liver, is intelligible. Rabbits, which have been treated with this monamine by intravenous injection and also by mouth for several weeks, developed aortitis and later on general arteriosclerosis and eventually nephritis.

The cellular elements of the blood are changed in their numerical relation after injections of this enterogenous monamine. There is polycythemia and lymphocytosis. When one reflects that these changes occur also after injections of adrenalin and constitute one of the blood signs of sympathicotonia, one cannot fail to be impressed with the close pharmacologic relationship between these substances. The reaction on the numerical relation of the blood cells does not occur when the hepatic artery is ligated in animals and the polycythemia is found in all the superficial veins of the animal and also in such human subjects that are afflicted with this Enterotoxaemia.

#### DEMONSTRATION OF P-HYDROXYPHENYLACETIC ACID IN THE URINE

Fifty cubic centimeters of the urine must be reduced to a specific gravity of 1015; 5 cc. of a 25 per cent solution of  $\text{H}_2\text{SO}_4$  is added; then follows extraction with 5 cc. of ether; this is allowed to evaporate, and thereafter, distilled water and Millon's reagent in equal parts are added. Millon's reagent is a solution of mercuric nitrate in water containing free nitrous acid. Any protein solution to which this is added produces first a white precipitate, which turns brick red color on boiling. In the urine of these patients, the reaction occurs in the following manner.

After repeated boiling, and allowing the urine to settle again, the amount, or degree of the coloring, is suggestive of the amount of the substance that gives the coloring matter. The colors may vary from pale red to dark red, maroon, or mahogany brown. Normal urinary constituents that give a red color with Millon's reagent, are insoluble in ether. Therefore, all urines which furnish an ether extract that gives a red or mahogany brown color with Millon's reagent, are pathological. The mother substance, which is the principal cause of these color changes, is tyrosine.

## BACTERIOLOGY

A bacillus has been isolated (J. J. Graham Brown) which belongs to the typhoid-coli group, a facultative-anaerobic bacillus which has the power to split up tyrosine and thereby form p-hydroxyphenyl-lethylamine.

## TREATMENT

It is essential that all meat diets be excluded; eggs and fish also. The diet should consist of foods made from flour, milk, sugar and butter. Some kinds of cheese can be allowed, especially cottage cheese, Edam cheese and cream cheese. Cheese which contains many bacteria, especially if it also presents dark spots, which are as a rule composed of tyrosine, should be excluded. Among these are Roquefort and Gorgonzola.

## INTESTINAL DISINFECTANTS

As all of these intestinal toxemias are cases of long standing before they are recognized as such, and as a rule have been treated for other conditions, or conceived of under another diagnosis, the treatment by chemical disinfectants would have to be continued for a long time, and there are no chemic substances which can be continued for months in the intestines without doing harm. The solution of urotropin or helmitol, one 10-grain tablet in a pint of hot water taken one hour before breakfast, is of utility but should not be kept up longer than two weeks. This solution can be given into any part of the bowel by the long intestinal tube, but the drinking is a much more practical method, and one which the patient prefers. Beta-naphthol, thymol, salol, have a period of usefulness, which, however, is limited, on account of the danger of these substances in producing nephritis, when continued a long time.

## COLON IRRIGATION

As the main seat of the production of enterotoxin is the colon, the washing out of the entire colon every day is one of the most effective means of preventing their formation. When systematic colon lavage is controlled by urinary examinations and bacterial cultures, it is found that the improvement is very evident after three weeks of treatment. The lavage should first be carried out with plain water. This should be followed by a colon irrigation with a 5 per cent solution of Isarol, of Ichthyol. If there is a tendency to diarrhea, the

colon irrigation should be performed with tannic acid, 40 to 60 grains to 1 quart of hot water. During diarrhea the diet should be strictly that adapted to this purpose; exclusion of liquids, fruits and vegetables, and the eating of a soup made of browned flour (see Hemmeter's Dietetic Kitchen).

#### AUTOGENOUS VACCINES

The processes which give rise to the formation of these monamines occur within the lumen of the intestine, that is, on the surface of the mucosa. Whether they occur in the actual tissue composing the intestinal wall, is extremely doubtful, because if they did so, the p-hydroxyphenylethylamine should be continued to be formed, even after the colon is washed out, and after the specific diet (proteins) from which these amines are formed are excluded; but it can easily be demonstrated, that under such conditions, the formation of these toxic monamines ceases. The process, therefore, is one that takes place in the lumen of the intestinal tract, and not in the walls.

This should be borne in mind in the treatment by auto-vaccine prepared under anaerobic conditions from the intestinal bacteria of the patient himself. It is very doubtful whether such a vaccine can produce effects, i.e., antibodies which shall check the growth of a specific intestinal flora in localities where the blood stream does not penetrate. This is my explanation of the frequent failure of the treatment of intestinal toxæmia by autogenous vaccines.

Another difficulty is that the isolation of the specific bacillus or streptococcus that is producing the dangerous monamine is even in the hands of the most skilled bacteriologist, a procedure of many trials and failures. Therefore, more dependence must be placed upon the diet, colon lavage and periodic use of intestinal disinfectants. If, however, the bacteriologist repeatedly reports that one and the same organism is continually predominant from the intestinal cultures, treatment by autogenous vaccines undoubtedly should be tried.

## THE DIAGNOSTIC AND THERAPEUTIC VALUE OF TRANSDUODENOBILIARY DRAINAGE

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In the evolution of new diagnostic or therapeutic methods there is always a time when their value may be overestimated because of undue enthusiasm on the part of those sponsoring them. On the other hand, there are those who, without satisfactory information, feel free to condemn as utterly worthless, methods that later are found to be of great value. Constructive criticism is always helpful in stimulating the worker along new lines to so perfect his work as to welcome the most careful investigation, but unmerited criticism tends to retard progress and favors reaction. It seems that all advancement must be made along these lines and those attempting to introduce new methods, which in their opinion are of great value to the profession, should not be discouraged if the results of their efforts are not unreservedly accepted at once. Nothing which has recently been brought to the attention of the profession would more definitely prove the above contention than the recent discussions relative to the value of duodenobiliary drainage as a diagnostic or therapeutic measure. In reporting the results of our somewhat limited experience with the method, which we have attempted for both diagnostic and therapeutic purposes, our only desire is to be of some assistance in determining a proper evaluation for the procedure which appears not yet to have emerged from its experimental stage.

The work of Doyon, Oddi and Meltzer forms a scientific background for the development of this procedure. Probably the work of Doyon (1) in 1894 was the first step of progress toward determining the intricacies of the functions of the bile passages and gall-bladder. As a result of his observation of the phenomena of the vagus and splanchnic stimulation, he was the first to call attention to the following: (a) That gall-bladder contraction with relaxation of the common duct sphincter was produced by stimulation of the central ends of the vagus; (b) that there was a definite interrelationship

(crossed innervation) of the bile passages, gall-bladder and common duct sphincter; (c) that irritation of the gastric and of the duodenal mucous membrane caused a relaxation of the common duct sphincter coincident with a possible contraction of the walls of the gall-bladder and bile ducts, as suggested by the increased discharge of bile; (d) that ammonia or vinegar could produce this relaxation. A little later, Oddi (2) showed that stimulation and reflex irritation of the nerve distribution of the first lumbar segment of the cord, produced a similar effect on the biliary tract, and because of his observations, the sphincter of the common bile duct at the neck of the ampulla of Vater received the appellation of "sphincter of Oddi."

In 1908-1909 Meltzer and Auer (3) published the results of their observations on the effect of magnesium salts on the central nervous system. They pointed out particularly the depressing effect of such solution on the peripheral nerve endings when applied directly to the duodenal mucous membrane. In their opinion, this brought about a relaxation of the mucous membrane, and of the Oddi sphincter, which was accompanied by a contraction of the gall-bladder and bile ducts, demonstrating the so-called physical law of contrary innervation. In 1917 Meltzer (4) further called attention to his observations that many substances (partially digested proteins, albumoses, peptones, hydrochloric acid) could produce limited demonstration of what he was pleased to call "the law of contrary innervation," and were probably the natural stimulants to the discharge of bile during the digestive phase. In a footnote, he stated that the local application of a 25 per cent solution of magnesium sulphate upon the mucosa caused a complete local relaxation of the intestinal wall; that the effect was not the same when administered by mouth; that the duodenal tube might be used for local application of the solution in cases of biliary colic and jaundice, in which cases it might relax the sphincter of the common duct and permit the ejection of bile and perhaps even the removal of a calculus of moderate size wedged in the duct in front of the papilla of Vater; that the procedure could be developed into a practical and useful method.

To Lyon (5, 6, 7, 8, 9, 10) must be given the credit for the early advocacy of the procedure of duodenobiliary drainage in a practical way. Working along the line suggested by Meltzer, he introduced a hyperisotonic solution of magnesium sulphate directly into the duodenum in the region of the ampulla of Vater by the duodenal tube and demonstrated that coincident with a relaxation of the duodenal

mucous membrane and probably of the sphincter of Oddi, there resulted an active discharge of fresh bile. His enthusiastic reports, supplemented by those of Smithies (11, 12), Friedenwald (13), White (14), Brown (15), Niles (16, 17), Sachs (18), Simon (19), Whipple (20), and others, have served to attract wide attention and to stimulate much individual study relative to the possibility of using this procedure in a diagnostic and therapeutic way.

#### RESPONSE OF BILIARY APPARATUS TO MAGNESIUM SALTS

The peculiar reaction on the part of the biliary mechanism to a hyperisotonic solution of magnesium sulphate when brought directly in contact for a brief period with the duodenal mucous membrane, is most interesting and has been described by Meltzer (4) as a similar phenomenon to the expulsion of the contents of the urinary bladder where with a relaxation of the sphincter, there is a coincident contraction of the musculature of the gall-bladder walls. This is questioned by some and particularly by White (14), who states: "The gall-bladder is a rather inactive, limp affair with little muscle fiber, and we are forcing the comparison a little when we compare it to the muscular urinary bladder. Even if the normal gall-bladder does contract after stimulation with magnesium sulphate, the thickened, rigid, fibrous, adherent, pathological ones do not." It is contended by some that a number of agents (various inorganic salt solutions, sodium sulphate, sodium phosphate, magnesium citrate (Einhorn 21, 22), weak solutions of hydrochloric acid (Bassler, 23)) act similarly to a solution of magnesium sulphate when applied to the duodenal mucosa, also that it acts equally as well when introduced into the stomach. These contentions in nowise detract from the practical value of the procedure, and while it may be demonstrated that other agents excite the Doyon-Meltzer reflex, so far it appears that magnesium sulphate is the most satisfactory and has a more specific effect when brought immediately in contact with the duodenal mucosa.

#### POSSIBLE SEGREGATION OF THE BILE FRACTIONS

Following the introduction of 50 to 75 cc. of a 33 $\frac{1}{3}$  per cent solution of magnesium sulphate into the duodenum, there results a free discharge of bile that can be collected in three characteristic fractions, designated by Lyon as "A," "B," and "C" bile; "A" bile normally about 25 to 30 cc., light golden yellow, supposedly of common duct

origin, "B" bile differentiated from the "A" bile by a decidedly darker color, dark golden brown, consisting of from 40 to 90 cc., supposedly of gall-bladder origin, and "C" bile, a lemon yellow somewhat lighter in color than that first collected and supposedly of hepatic duct origin. The designation of these fractions as definitely coming from the different portions of the biliary apparatus has seemed rather fanciful to some and has aroused much discussion pro and con, but the sequence of the distinct and characteristic "A," "B," and "C" bile fractions, following the introduction of magnesium sulphate solution into the duodenum, cannot be denied.

While the segregation of the bile has not been proved to the satisfaction of all, we would suggest the following explanation which may be somewhat theoretical but which is apparently substantiated by our observations supplemented by the results reported by a number of others. The bile normally found in the common duct during the interdigestive or fasting stage is probably liver bile somewhat changed by the length of time it remains in the common duct. In appearance it corresponds quite closely to that obtained at the close of a biliary drainage and has probably never been stored in the gall-bladder. When there is a demand for bile during the digestive period, resulting from stimulation of the duodenal mucous membrane by the acid chyme, as suggested by Pawlow and Matthews, or by partially digested protein substances, according to Starling, Meltzer and others, there results a discharge of the accumulated bile which corresponds to the "A" bile. If the stimulation is mild, there probably continues a type of bile of light golden yellow such as we observe when we introduce a drainage tube into the duodenum and collect the discharged bile. In such cases we do not obtain any dark bile. If we then lavage the duodenal mucous membrane with magnesium sulphate solution, we obtain a dark fraction, the amount and color of which depend somewhat on the time of application and the strength of the solution used.

#### SOURCE OF "B" BILE

It is around the source of this dark fraction that there has been so much discussion and on which depends in a large measure the value of the procedure. It has been shown by the published work of Rous and McMaster (26, 27), that the function of the gall-bladder is in all probability for the purpose of concentrating and storing the excess of bile over the immediate digestive needs, and on account of its small

size, this can only be done by concentrating the volume received. They have demonstrated that in certain animals, the bile may be concentrated as high as ten times in a reasonably short period of time and that the change in color is probably due to the concentration of the pigment incident to the absorption of water by the gall-bladder mucosa. Pawlow's assistants called attention to the fact that they recognized the discharge of the gall-bladder bile during the digestive phase in dogs on account of its increased color, and certainly the work of these men would indicate that the function of the gall-bladder was to store the bile during the interdigestive phase with a discharge during the digestive period incident to the stimulation of partially digested proteins in the acid chyme from the stomach. Pointing out the possibility of the formation of calculi in the gall-bladder as a result of stasis, they (as did Meltzer in 1917) have suggested that this could be avoided by the frequent introduction of foodstuffs, and that the taking of meals at rare intervals or a period of fasting was a questionable procedure, as it decidedly favored biliary stasis and the formation of calculi in the inspissated gall-bladder bile.

The possibility of the darker colored fraction resulting from an oxidation process on the part of the liver cells, as suggested by Bassler (23), Einhorn (22) and Kohn (24), seems highly improbable from our observations for the following reasons: (*a*) We have been unable to obtain this in cases having a previous cholecystectomy. We have carefully studied our post-operative cases at different periods from a few days to a year or more, but at no time did we find it possible to obtain a dark fraction. (*b*) We have not obtained it in any case where later at operation, it has been found that the cystic duct was obstructed by calculi, adhesions, new growths, or extreme inflammatory swelling. (*c*) In certain cases where repeated attempts to obtain a dark fraction had resulted in failure, it was found at operation that the gall-bladder was so densely packed with stones as to have completely lost its function as a biliary receptacle, while in others it was found to be so completely atrophied, probably as a result of long continued inflammation with the formation of extensive adhesions, as to be practically obliterated.

If the magnesium sulphate solution produces a dark colored fraction by its action on the liver, it should be as readily obtained in the absence of the gall-bladder, but this is contrary to our observations. The apparent exception to this, as reported by Dunn and Connell (25), might suggest a stasis of bile in some part of the biliary mechan-

ism, due to dilatation of the ducts or to disturbed hepatic function incident to extensive pathology as may be observed after the extirpation of a diseased gall-bladder. During a period when food is taken sparingly if at all, it has been observed by Rous and McMaster (26) that there is a secretion of a smaller quantity of bile of higher pigment concentration. Further, the very rare exceptions as reported in literature of cases where bile is obtained after cholecystectomy would appear of itself to substantiate the idea that in some way the gall-bladder is essential to the dark fraction. In certain pathological conditions it may be possible, as suggested by Rous and McMaster, that a dark bile might be produced in the liver which simulates in appearance that ordinarily found in the gall-bladder, but it is under extremely abnormal conditions.

The degree of color of "B" bile we believe results from (a) the character of the bile stored in the gall-bladder at the immediate time of observation, which depends on a number of factors such as the functional condition of the liver, the diet, frequency of meals, presence of infection, presence of stasis, etc., and (b) the degree of stimulation applied to the duodenal mucous membrane and the length of time the stimulant remains in contact with the duodenal mucosa, particularly in the vicinity of the ampulla of Vater. In our own experience we have observed a number of cases where we were unable to obtain the "B" bile on the occasion of our first test, whereas after repeated efforts we would obtain a very satisfactory specimen. In all probability our failure was due to our not being able to apply the magnesium sulphate in proper degree of concentration to that portion of the duodenal mucous membrane which controls the relaxation of Oddi's sphincter and reflexly stimulates the biliary tract to discharge its bile. This would suggest the danger of drawing definite conclusions as to a possible obstruction of the cystic duct or other definite pathology at some portion of the biliary tract, as the result of observations made at one single drainage.

The presence of a Rehfuß bulb in the region of the ampulla of Vater apparently causes biliary incontinence, as we have obtained as high as 300 cc. of lemon colored bile during a five hour period. This might explain why Crohn's (28) experience was not more satisfactory inasmuch as in some cases he introduced the tube the night before. The gall-bladder bile is no doubt a reserve which is used only when sufficient bile is not produced by the liver cells to properly care for the immediate digestive needs. The amount of the dark bile obtained

after stimulation by magnesium sulphate solution has been considered an indication of gall-bladder capacity and suggestive of abnormal distention when the amount exceeded 75 cc. (Smithies, 12) or 90 cc. (Friedenwald, 13). The quantity obtained would seem to us to depend somewhat on other factors. In comparing the amount obtained at different times with the same individual, we find a decided variation. Moreover, we have at times been able to obtain a further discharge of dark bile after the flow had assumed the lemon yellow color supposedly of liver origin. Possibly in the majority of cases there is a complete emptying of the gall-bladder but this we feel depends somewhat on the degree of concentration and the time of application of the magnesium sulphate solution.

Further, relative to the source of the dark fraction: In comparing the results obtained at duodenobiliary drainage in suspicious cases of biliary disease with those obtained from a series of cases where the subjects were young individuals in apparently perfect health, without history of previous infections predisposing to biliary infection, we find a decided contrast. In our normal cases the "B" bile was golden brown, clear, slightly viscous and free from mucus, while in cases showing definite clinical evidence of gall-bladder disturbance, the "B" bile was very dark greenish-brown, at times almost an inky black, extremely viscous, with or without strings or shreds of mucus. In certain of these cases referred for operation, the gall-bladder was found to contain bile typical of that obtained at previous duodenobiliary drainage. This is in accord with the observations of Rous and McMaster whose deductions are to the effect that the bile becomes "heavily pigmented, syrupy, ropy, or even tarry" depending on the period of obstruction or stasis. The frequent therapeutic drainage of similar observed cases over a considerable period of time brought about a decided change in the color and character of the "B" bile such as might be expected where stasis was removed. Crohn's (28) observations along this line are very interesting. In reporting on twenty cases followed to operation he stated that 65 per cent gave "a dark brown or greenish-black bile" after douching with magnesium sulphate, and that 62 per cent of these cases had definite pathology (stones and chronic cholecystitis) while the remaining 38 per cent were found to be normal.

## TECHNIQUE OF METHOD

The method followed in our work is somewhat similar to that suggested by Lyon. The patient appears, not having taken food for a period of twelve hours. The mouth and throat receive careful attention, every effort being made to free them as nearly as possible from contaminating organisms. We use the Einhorn or Rehfuess tube which is sterilized according to surgical asepsis. In a part of our cases we have attempted to thoroughly lavage the stomach until the aspirate became clear, hoping that we could avoid contamination from this source. We have not felt that we were entirely successful in this respect, as the cases so treated failed to show any material change in the percentage of infection in the duodenal contents and the biliary drainage. We further find that the passage of the tube into the duodenum seems to be accomplished with more difficulty after this procedure. The location of the tube is demonstrated by the presence of an alkaline fluid, usually bile-stained, and in the majority of cases, it is also determined by roentgen ray observation. The magnesium sulphate solution is introduced slowly either by gravity or by very gentle pressure on the syringe. It is allowed to remain in the duodenum three to eight minutes before being siphoned off. In the majority of cases the bulb enters the duodenum with very little delay, allowing the entire drainage to be completed in from one and one-half to two hours. In other cases, particularly where the patient is in an apprehensive state of mind, the introduction of the tube is found to be impossible. Such cases may be successfully treated a day or two later after they have lost their apprehension and are in a relaxed condition, or it may be helpful to resort to the use of atropine, benzyl benzoate, or a mild sedative. In certain cases where the duodenal tube is in place as determined by the roentgen ray and where it is possible to obtain the "A" and "C" bile, it seems impossible to obtain the "B" bile even after repeated douching with magnesium sulphate solution. Just why this is so, it is difficult to explain, particularly where there are no clinical indications of an obstructed cystic duct or other serious pathology affecting the gall-bladder. Frequently, by repeating the drainage at two or three day intervals, the second or third attempt may prove to be entirely successful.

## PHYSICAL CHARACTERISTICS

The quantity obtained supposedly from the three different locations, common duct, gall-bladder and hepatic ducts may vary considerably. The normal average in our cases was 22 cc. of the "A" specimen, 37 cc. of the "B" specimen, and the amount of "C" depended on the time drainage was continued. Marked deviations from these amounts are no doubt significant. We have not found the marked deviations in quantity of "B" bile as reported by others; there has rarely been over 100 cc. Biliary stasis cannot be determined, in our opinion, by amounts in excess of 90 cc. as suggested by Friedenwald (13), as in cases where the gall-bladder is extremely dilated, there may be some definite obstruction which prevents it from completely discharging its bile in response to the stimulation of the magnesium sulphate solution, or there may be definite atony of the gall-bladder, and in probably a majority of cases found definitely diseased by the surgeon, either with or without stones, the gall-bladder may be contracted. Therefore, a small amount of black bile may be more significant of stasis than a larger amount.

The fractions obtained from suspected cases frequently contain clots or strings of mucus, or mucopurulent material, in marked contrast to those obtained from our normals, which were uniformly clear, almost transparent. While a cloudiness may be produced by the admixture of stomach fluid, this can usually be detected at once by the operator and should not be confused with mucus of duodenal or biliary tract origin. In all cases where there is a definite turbidity of any of the three fractions, we believe that it suggests an abnormal condition. The presence of mucus in macroscopical amount is probably abnormal. Where it occurs in all three fractions, it probably is indicative of a duodenitis but if found only in "B" bile or "C" bile it is at least suggestive of a choledochitis or a possible cholecystitis of a catarrhal character.

We have carefully observed the specific gravity of our "A," "B," and "C," fractions and have noted wide variations particularly with the "A" bile. Inasmuch as "A" is apt to be mixed with a certain amount of magnesium sulphate solution, the specific gravity of this fraction is not dependable. If the collection of the specimen is delayed until this solution has all been aspirated, or has passed downward in the intestine, a considerable amount of the so-called common duct bile has been lost. The average specific gravity of our specimens was as follows: "A" bile 1.030, "B" bile 1.020, "C" bile 1.019.

Marked deviations from these, where particular care has been taken to avoid admixture of gastric contents or the magnesium sulphate solution would be suggestive of pathology.

#### POSSIBLE FUTURE METHOD FOR ESTIMATION OF PANCREATIC ACTIVITY

Up to the time of the development of aspiration of duodenal contents, we have had no reliable method for determining the activity of the pancreatic ferments. A study of their activity in the material obtained at duodenobiliary drainage would seem to be a satisfactory procedure for determining the functional condition of this most important gland of the digestive system, and also a reliable method for observing deviations from the normal, which may enable us to more accurately diagnose pancreatic disorders. Whether this procedure is as satisfactory as some of the test meals for the determination of pancreatic activities is questionable, but we feel that it warrants further comparative study.

#### BACTERIOLOGICAL OBSERVATIONS

Relative to the bacteriological findings, we are at present unable to speak with the enthusiasm and apparent authority of certain observers who have largely based their diagnostic conclusions on the presence of certain types of bacteria found in one or more of the fractions collected. It has been considered that the stomach and duodenum were normally free from bacteria but this is contrary to our findings. As the majority of our patients suffer from some type of upper respiratory infections, it is probable that the stomach and also duodenal contents are quite commonly infected from this source, as would seem to be proven by our observations. It is impossible to believe that our efforts to free these localities of infection are entirely successful. Where the stomach contents are infected, we are apt to find a decided similarity in the duodenal flora and this may persist in the "A," "B," and "C" bile fractions.

It is difficult to believe that it is possible to definitely locate the source of infection found in the duodenal aspirate. If the source of this infection is in the mouth or upper respiratory tract, we could easily imagine the infection being carried to the stomach and thence to the duodenum where it might remain for sufficient time even with every effort to free the stomach from infection, to contaminate the

bile fractions to a greater or less degree. It is explained that it is the degree of infection that is to determine the locality infected, which seems reasonable but difficult to determine. Moreover, by comparing the repeated bacteriological studies on the same individual, we find a *B. coli* infection on one occasion, a streptococcic a few days

TABLE 1  
*Bacteriological findings of "A," "B," and "C" bile*  
Total number of cases 400

|   | "A" BILE        |          | "B" BILE        |          | "C" BILE        |          |
|---|-----------------|----------|-----------------|----------|-----------------|----------|
|   | Number of cases | Per cent | Number of cases | Per cent | Number of cases | Per cent |
| Aspirate obtained . . . . .                                   | 333             | 83       | 327             | 82       | 268             | 67       |
| Infected . . . . .  | 233             | 70       | 182             | 56       | 122             | 45.5     |
| Sterile . . . . .   | 100             | 30       | 145             | 44       | 146             | 54.5     |
| Infected with 1 organism . . . . .                            | 162             | 70       | 138             | 70       | 88              | 72       |
| Infected with 2 organisms . . . . .                           | 66              | 28       | 36              | 20       | 28              | 23       |
| Infected with 3 organisms . . . . .                           | 17              | 7        | 8               | 4.5      | 6               | 5        |
| Infected with more than 3 organisms . . . . .                 | 2               |          | 0               |          | 0               |          |
| Infected with staphylococci . . . . .                         | 131             | 56       | 104             | 57       | 72              | 59       |
| Infected with <i>B. coli</i> . . . . .                        | 69              | 29       | 62              | 34       | 37              | 30       |
| Infected with yeast . . . . .                                 | 75              | 32       | 56              | 31       | 28              | 23       |
| Infected with streptococci . . . . .                          | 54              | 23       | 37              | 20       | 23              | 19       |
| Infected with <i>B. proteus</i> . . . . .                     | 43              | 18       | 40              | 22       | 27              | 22       |
| Infected with <i>B. subtilis</i> . . . . .                    | 20              | 9        | 17              | 9        | 8               | 6.5      |
| Infected with gram positive bacteria (unidentified) . . . . . | 20              | 9        | 12              | 7        | 11              | 9        |
| Infected with gram negative bacteria (unidentified) . . . . . |                 |          | 4               |          | 3               |          |
| Infected with <i>B. welchi</i> . . . . .                      | 8               |          | 2               |          | 2               |          |
| Infected with acidophilus . . . . .                           |                 |          | 1               |          |                 |          |
| Infected with diphtheroids . . . . .                          |                 |          |                 |          | 1               |          |

later and possibly a staphylococcic on a subsequent study. As a result of our studies on 400 cases (see tables) we found the stomach infected in 82 per cent, "A" bile in 70 per cent, "B" bile in 56 per cent and "C" bile in 54.5 per cent. The streptococcus was found in 58 per cent of cases studied, the staphylococcus in 50 per cent and *B. coli* in 15 per cent. In the bile fractions, the order of frequency was different, the staphylococcus being found in the "A," "B," and

TABLE 2  
*Bacteriological findings of "B" bile in clinically normal cases*

| CASE NUMBER | DATE OF DRAINAGE | BACTERIA CULTURED                       | REMARKS   |
|-------------|------------------|---|---|
| 2-J-115     | 3/ 8/22          | B. coli                                 | B. coli also in "C" bile<br>"A" and "C" bile infected<br>"A" and "C" also contain staphylococci.                          |
|             | 3/14/22          | Sterile                                 |   |
|             | 3/20/22          | Staphylococci                           |   |
| 2-J-113     | 3/ 8/22          | Sterile                                 | "A" bile contains B. coli<br>"A" bile contains staphylococci<br>"A" and "C" markedly infected                             |
|             | 3/14/22          | Staphylococci                           |   |
|             | 3/26/22          | B. coli (few)                           |   |
| 1-J-217     | 4/29/21          | Streptococci                            | "A" and "C" also contain streptococci   |
|             | 5/ 6/21          | Sterile                                 |   |
| 1-J-218     | 4/29/21          | Sterile                                 |   |
|             | 5/ 6/21          | Sterile                                 |   |
| 1-J-200     | 4/29/21          | Staphylococci                           | "A" and "C" bile infected   |
|             | 5/ 6/21          | Sterile                                 |   |
| 1-J-215     | 4/ 4/21          | Staphylococci (only)                    | Same in "A" and "C" bile<br><br>Same in "A" and "C" bile  |
|             | 5/ 5/21          | Staphylococci, streptococci and B. coli |   |
| 1-J-192     | 4/26/21          | Sterile                                 |   |
|             | 5 /5/21          | Sterile                                 |   |
| 1-J 234     | 4/13/21          | Sterile                                 | Same in "A" and "C" bile  |
|             | 6/16/21          | Sarcinae and B. subtilis                |   |
| H. R.       | 4/ 5/21          | Sterile                                 | "A" bile contains streptococci and staphylococci<br>"C" bile contains staphylococci<br>"A" and "C" contains staphylococci |
| 1-J-202     |                  | Sterile                                 |   |
| Miss F.     |                  | Sterile                                 |   |
| 1-J-195     |                  | Sterile                                 |   |
| 1-J-378     |                  | Staphylococci                           |   |
| Miss. G.B.  |                  | Sterile                                 |   |

*Summary of normal cases (24 drainages): "B" bile infected in ten or 42 per cent; sterile in 14 or 58 per cent; and sterile "A," "B," and "C" fractions in 9 or 37 per cent.*

TABLE 3  
*Bacteriological findings of "B" bile in clinically pathological cases*

| CASE NUMBER | DATE OF DRAINAGE | BACTERIA CULTURED                        | REMARKS   |
|-------------|------------------|--|---|
| 1-J-363     | 1/18/21          | Sterile                                  | "A" bile contains staphylococci                           |
|             | 8/23/21          | Sterile                                  |   |
|             | 1/18/22          | Diphtheroids                             | "A" and "C" markedly infected                             |
|             | 1/30/22          | Staphylococci                            | "A" and "C" infected                                      |
| 1-J-203     | 4/10/21          | Sterile                                  | "C" bile infected   |
|             | 5/ 2/21          | Sterile                                  |   |
|             | 6/ 6/21          | B. coli                                  | "A" and "C" contains B. coli                              |
|             | 7/11/21          | Staphylococci                            | "A" contains B. coli                                      |
|             | 8/ 8/21          | Sterile                                  | "C" contains B. coli                                      |
| 2-J-5       | 11/22/20         | B. coli                                  | Same in "A" and "C"                                       |
|             | 12/31/20         | Sterile                                  |   |
|             | 2/ 1/21          | Staphylococci and B. proteus             | Same in "A" bile  |
|             | 3/31/21          | Sterile                                  |   |
|             | 1/ 2/22          | Diphtheroids and Streptococci            | Staphylococci, Streptococci, and diphtheroids in "C" bile |
| 1-J-265     | 12/31/20         | B. welchii                               | Same in "A" and "C" bile                                  |
|             | 1/30/21          | B. welchii                               | Same in "A" and "C" bile                                  |
|             | 2/28/21          | B. proteus                               | Same in "A" and "C" bile                                  |
|             | 5/26/21          | B. coli                                  | "A" bile infected   |
|             | 3/ 9/22          | B. proteus                               | "A" and "C" bile contain B. proteus and B. coli           |
| 1-J-109     | 10/31/21         | B. welchii, streptococci, yeast          | "A" bile contains staphylococci and streptococci          |
|             | 11/25/21         | Staphylococci                            | "A" and "C" bile infected                                 |
|             | 2/ 1/22          | Staphylococci and B. coli                | "A" and "C" bile infected                                 |
|             | 3/ 9/22          | Diphtheroids                             | "A" and "C" contain B. coli                               |
| 2-J-48      | 12/ 7/21         | Staphylococci streptococci, diphtheroids | "A" and "C" bile infected                                 |
|             | 12/27/21         | Staphylococci, streptococci, B. coli     | "A" and "C" same  |
|             | 1/27/22          | Staphylococci yeast, diphtheroids        | "A" and "C" bile infected                                 |
| 2-J-55      | 2/ 2/22          | B. proteus                               | Same in "A" and "C" bile                                  |
|             | 2/ 5/22          | B. coli and staphylococci                | Same in "A" and "C" bile                                  |
|             | 2/13/22          | B. coli and staphylococci                | Same in "A" and "C" bile                                  |

*Summary (29 drainages).* "B" bile infected in 22 or 76 per cent; sterile in 7 or 24 per cent.

"C" bile in 56, 57, and 59 per cent respectively, the streptococcus in 23, 20, and 19 per cent, while the *B. coli* was found in 69, 34 and 30 per cent. It is possible that colon bacillus infection of the upper

TABLE 4  
*Bacteriological findings of "B" bile in surgical cases*

| CASE NUMBER | DATE OF DRAINAGE  | BACTERIA CULTURED   | REMARKS   |
|-------------|---|---|---|
| Mrs. A.     | 1/12/21<br>1/17/21  | <i>B. coli</i><br>Sterile   | "A" and "C" contain <i>B. welchi</i><br>"C" bile contains staphylococci<br><i>Operative findings:</i> many gall-stones  |
| 2-J-141     | 3/23/22<br>4/23/22  | <i>B. coli</i> , diphtheroids<br>Unable to obtain "B" bile  | "A" and "C" bile infected<br><br><i>Operative findings:</i> 19 gall stones  |
| 1-J-347     | 8/ 5/21   | <i>B. coli</i>  | "A" contains staphylococci and streptococci.<br><i>Operative findings:</i> 40 gall stones   |
| 1-J-523     | 12/28/21  | Staphylococci   | "A" and "C" infected<br><i>Operative findings:</i> many gall stones   |
| 2-J-12      | 1/ 7/21<br>2/ 4/21<br>2/10/21<br>2/14/21<br>4/25/21<br>10/31/21<br>1/ 7/22<br>1/26/22 | Staphylococci and diphtheroids<br>Staphylococci<br>Streptococci and <i>B. welchii</i><br><i>B. coli</i><br>Staphylococci<br>Yeast<br>Staphylococci and diphtheroids<br>Staphylococci and streptococci | "A" and "C" bile infected<br>"A" and "C" same<br>"A" bile same<br>"A" bile streptococci and <i>B. coli</i><br>"A" bile infected<br>"A" and "C" infected<br>"A" and "C" infected<br>"A" and "C" infected<br><br><i>Operative findings:</i> strawberry gall-bladder and cirrhosis of liver. |

digestive tract is abnormal and may suggest, when found in the biliary drainages, disease of some part of the tract, but this has not been proved by our observations. While in some cases a definite simi-

larity has been observed between the bacteriological findings in the bile that has been removed aseptically at operation and that obtained by duodenobiliary drainage, in the majority of cases this has not been apparent. Furthermore, if biliary infections are blood borne (Rose-now, 29), it is possible that the infective agent may be confined to the mucosa and submucosa, and not necessarily found in the contents of the gall-bladder or bile ducts. While we are not willing to conclude from our studies that the information obtained by bacteriological observation is necessarily of small value diagnostically, we do feel that further careful study is necessary before we are able to draw definite conclusions.

#### CYTOLOGICAL STUDY

Relative to our microscopical and cytological findings, we are again somewhat disappointed in not being able to report them as definitely helpful in a diagnostic way. Lyon suggests that any considerable time elapsing between the removal of the bile and the microscopical examination allows for the digestion of the cells. We have definitely corroborated this. We have not found in cases of surgically proved gall-bladder disease where gall stones have apparently been harbored for long periods, that we were able to demonstrate pus cells in the specimens obtained. Epithelial cells are found in considerable frequency as are desquamated goblet cells. Since the bile ducts, gall-bladder and also the duodenum, are lined with columnar epithelium, we confess to embarrassment in attempting to differentiate as to their source, or to base pathological interpretations on their presence. We would therefore be loath to place great importance as a diagnostic help on the presence or absence of pus cells, or on the particular type of epithelium found. The presence of duodenal epithelium in abnormal amounts may be suggestive of duodenal pathology. In addition to the finding of epithelial cells and bacteria, the constant finding of amorphous bile salts, fatty acid crystals, and cholesterin crystals has attracted our attention but we have not been able to utilize this information in a definitely diagnostic way. We have found cholesterin crystals in both pathological and non-pathological cases, as well as in the "B" bile and in the bile obtained from the same patient a week following cholecystectomy. Therefore, we hesitate to conclude that their presence is suggestive of a precalculous state. The findings of parasites as reported by certain observers is interesting but so rare as to be of little signifi-

cance. It may be that further study along these lines will be rewarded by more definite information.

#### DIAGNOSIS

The findings obtained from a study of duodenobiliary drainage in cases of obscure digestive disturbances, where our previous methods have failed to permit of a positive diagnosis, we believe to be very helpful in definitely localizing the source of trouble and in many cases obviates the necessity of surgical exploration for diagnostic purposes. There was a period in the evolution of the examination of gastric contents, following the test meal, when a diagnosis was supposedly possible on the basis of such findings only. We now recognize that this was unfortunate and that to avoid error the findings obtained from gastric analysis had to be considered in connection with evidence obtained from other sources in order to be of valuable diagnostic help. So with the information obtained by duodenobiliary drainage, we may be led into error if we attempt a diagnosis on the basis of this alone. An abnormally dark "B" bile suggests stasis but this may be a temporary condition incident to disturbances in other parts of the gastrointestinal tract, the absence of proteins in the diet, fasting, etc., and might not alone warrant a diagnosis of gall-bladder pathology. Nor do we believe that the finding of some definite type of bacteria in a certain fraction would necessarily warrant a definite infection of that locality. If this were the case, we would be compelled from our findings to conclude that a large proportion of the patients we observe suffer from biliary tract infection which we are not convinced is the case. It is only when there are, in addition to these findings, clinical manifestations of trouble with a very suggestive history in addition to X-ray and physical findings, that we would be justified in making a positive diagnosis. We have not been so successful with the method in a diagnostic way as has Lyon, who, in reporting 100 cases of gall-bladder disease, states: "Thirty-two of them could have been readily diagnosed as gall tract disease if studied only in the light of the history and physical examination, supplemented by roentgen ray examination and by our usual gastrointestinal methods; whereas 68 per cent of them would have failed of such diagnosis except by means of direct cytology, bacteriology and chemistry of the bile." However, as a result of our experience with the method, we believe it to be of such definite diagnostic assistance as to warrant classifying it among other accepted procedures as an aid in the diagnosis of right upper quadrant lesions.

## THERAPEUTIC POSSIBILITIES

The therapeutic possibilities of transduodenobiliary drainage are attracting much favorable attention and some adverse criticism. There is no problem of greater importance before the medical profession today than that of proper therapy in right upper quadrant disorders, particularly those of the biliary tract. The recent tendency has been to invoke surgical aid, not only to clear up a doubtful diagnosis, but also for therapeutic purposes where there was any doubt as to the results of non-surgical treatment. The frequency in which cholecystectomy is employed for the relief of vague digestive disturbances where the macroscopical examination of the gall-bladder at time of operation does not definitely indicate pathology, and the many instances where the results are unsatisfactory, if not unfortunate, might warrant the suspicion that we have been overzealous in our reliance on surgical therapy for the relief of such conditions. If non-surgical drainage can be employed successfully in the treatment as well as in the diagnosis of this relatively large class of vague abdominal disturbances, we cannot with propriety, cast it aside.

Our original purpose in employing the method was not for therapeutic reasons but rather for diagnostic purposes only. However, our patients so frequently reported temporary relief from their symptoms and in so many instances requested a repetition of the procedure, that we were soon forced to the conclusion that the relief experienced was more than a psychic effect, since we never received requests for repetition of the fractional gastric test which is a similar process as experienced by the patient. We have employed the procedure, as a therapeutic measure and believe it to be of decided benefit in suspicious biliary tract disorders where it has not been possible by the generally accepted methods of examination, to arrive at a diagnosis of definite pathology necessitating surgical intervention. Our experience may be based upon insufficient evidence, and we may be laying ourselves liable to criticism in using a method of unproved value, but the history of medical therapeutics is not entirely free from a suspicion of empiricism. There is always a period when the results obtained must be the criterion supporting its use rather than our ability to scientifically explain the results.

There may be a question in the minds of some as to the possibility of relieving permanently an infected biliary tract by duodenal drainage. We may not be in a position to prove that this is possible, neither have we definite proof that the body is unable to eradicate an

infection in the gall-bladder as has been proved in cases of infection in other localities. We believe that chronic cholecystitis is similar to chronic appendicitis and that there is probably a disease of the whole biliary tract; hence, the removal of the gall-bladder does not any more thoroughly relieve the disturbance than does the diseased appendix when the entire colon is involved. We have definitely proved at operation, even where gall stones have been present for an indefinite time, that the bile is sterile and that the symptoms requiring surgical interference probably resulted from mechanical irritation rather than from toxemia of infectious origin. If this is true, then it would seem probable that at some time prior to the formation of the calculi the relief of biliary stasis by proper therapeutic procedure might have prevented the formation of stones, obviating the necessity for surgery at a later time. It is possible that the duodenobiliary drainage is only one of many measures to be considered in accomplishing this. Its most valuable use may be to reveal a condition favoring gall stone formation, the relief to be obtained by other means; regulation of physical and dietetic habits and such corrective measures as the removal of definite foci of infection in diseased tonsils, infected teeth, etc.

#### CASES INDICATING TRANSDUODENOBILIARY DRAINAGE FOR THERAPEUTIC PURPOSES

There are two general classes of cases where we have found this procedure to be decidedly beneficial as a therapeutic measure: (a) Cases where the clinical symptoms were suggestive of biliary stasis with possible mild infection, but where the evidence was not sufficiently positive to warrant surgical interference. These patients exhibited vague digestive symptoms apparently not of definite gastrointestinal origin, some giving a history of migraine, others having the more persistent type of headache apparently of toxic origin, while still others showed definite symptoms of acute catarrhal jaundice and a chronic jaundice suggestive of biliary sclerosis. (b) Cases showing definite evidence of biliary tract pathology but where it was inadvisable to recommend surgery on account of other complications such as degenerative myocarditis, angina pectoris, hypertension, etc.; also cases where surgery was advised but refused for personal reasons. In this latter group the drainage was used for the temporary relief of distressing symptoms rather than for permanent relief.

Our results with the first group have so far been decidedly encouraging. In the institutional treatment of patients suffering from a more or less chronic condition, several lines of therapeutic endeavor may be used simultaneously (correction of unfortunate dietetic habits, the use of hydrotherapy, massage, rest, graduated exercises, etc.) so that at times it is difficult to accurately determine the definite value of any one of the measures employed. But in these cases giving definite clinical symptoms of chronic gall-bladder infection, and where the physical, chemical and possibly the bacteriological findings of fractions obtained by duodenobiliary drainage are all corroborative, very marked improvement has been noted following repeated drainages. The gradual approach to normal in physical and chemical appearance of the fractions, with coincident improvement in the patient's clinical condition, has been observed frequently in our studies, corroborating the experiences of Lyon, Smithies, and others. In the use of this new procedure, we have tried to avoid the mistake of ascribing to it undue credit, comparing the treated cases with others under similar conditions with the exception of drainage.

In our migraine cases which exhibit definite indications of biliary stasis, there has been a decided amelioration of symptoms following repeated duodenobiliary drainage. The attacks became less frequent and less severe; the nausea and vomiting in most of the treated cases was entirely relieved; there was an improvement in appetite and a decided gain in weight; as a result of which we feel that we are warranted in the conclusion that biliary stasis and possibly infection, is a frequent finding in migraine and that the relief of the stasis results in prompt improvement in the patient's condition. Very frequently these patients report relief following the use of cathartics and saline laxatives, which is possibly due to the relief of the biliary stasis rather than an intestinal stasis.

In the majority of cases where we have felt that duodenobiliary drainage was indicated, its use has apparently resulted in at least temporary relief to the patient. The immediate feeling of well being, the improvement in appetite and general nutrition is so apparent to the patient himself as to cause him to insist upon continuing the treatment at intervals, even at great personal inconvenience. In some instances the patient with the assistance of some member of his family, continues the treatment after his return home with apparently as good results as when administered by a trained attendant. The marked change in the physical appearance of bile after continued

treatment, warrants us in believing that it is possible to bring about such changes as will permit of its approximating the normal in this respect at least.

#### CONCLUSIONS

Extravagant and unwarranted claims as to the value of the procedure, either diagnostically or therapeutically, at the present time we feel would be unfortunate. Our experience would warrant the belief that it can be used with decided advantage in selected cases, and in time will no doubt take its place among other proved methods to the credit of those who have been instrumental in introducing it to the profession through their carefully recorded and painstaking observations. We hope that with greater experience it will be possible for us to report as enthusiastically as some of our confrères. We are inspired by their great success to press on, hoping that it may be of such value in our hands as they relate.

#### SUMMARY

Our experience with the method for somewhat over a year warrants the following conclusions as a result of our observations:

1. The possibility of segregating the bile from different parts of the biliary mechanism by the application of a magnesium sulphate solution to the duodenal mucous membrane so that the specimens obtained are reasonably typical of common duct, gall-bladder, and hepatic duct bile, has been demonstrated to our satisfaction.

2. A careful study of the physical characteristics of the fractions obtained from suspected cases and their comparison with those obtained from normals or unsuspected cases used as controls, gives valuable information as suggested by the degree of color, viscosity, specific gravity, and amount of "B" bile, while the presence of mucous shreds and chunks of muco-purulent material probably indicated biliary tract pathology.

3. A careful review of our bacteriological findings would not warrant us in believing that the information obtained in this way is of such value as to permit of its being entitled to very great consideration. Lack of uniformity in our findings without change in clinical symptoms, suggests unavoidable bacteriological contamination rendering the findings unreliable.

4. We are also unable to attach any great importance to our cytological findings. The definite localization of an infection by deter-

mining the presence of pus, blood or certain types of epithelial cells, we believe is practically impossible.

5. The value of the method as a diagnostic procedure, in spite of our failure along bacteriological and cytological lines, warrants our continuing its use. It has legitimate therapeutic possibilities in selected cases. The relief of biliary stasis not only gives temporary relief but may be a valuable aid in restoring the individual to a normal condition.

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## DISCUSSION

DR. JOHN C. HEMMETER, BALTIMORE: Dr. Eggleston said that he did not know who assumed the parentage of this method. If he will look up the Johns Hopkins Hospital Bulletin for April, 1895, he will find that Dr. Hemmeter does. I shall have to assume the parentage of it, as I brought it before the society and was then on the editorial Staff of the Archiv f. Verdauungskrankheiten Berlin, where I published the later articles. I saw a man yesterday whom I treated by non-surgical biliary drainage twenty-two years ago. I think that it is more instructive and reliable to judge of the remote effects on a few patients treated a number of years ago than on many patients treated only recently, that is not over a month or six months ago. I have patients who were treated at different times. A certain member claims that he was the author of the method and that no one used it but him. Concerning this claim I refer to my article on the history of the discovery of duodenal intubation (N. Y. Medical Record, February 26, 1921). I am glad to be the father of a child which has produced such wide interest and enthusiasm as did this method among the clinicians applying it.

I do not think that the differences between Dr. Eggleston and Dr. Lyon are irreconcilable. If Dr. Eggleston could see Dr. Lyon work in his own laboratory as I have seen him, he would become convinced that essentially his statements are correct. I have been at his workshop a number of times, and have worked with him.

*Concerning reciprocal or contrary innervation between the gall-bladder and the sphincter at the papilla of Vater (sphincter of Oddi):* This reciprocal innervation of the gall-bladder, the ducts and the sphincter of Oddi was not first studied by Meltzer but by Doyon (Archives de Physiologie, 1894). "It would seem, from the experiments made by this author together with later experiments reported by others, that the bladder receives both motor and inhibitory fibers by way of the splanchnic nerves. These fibers emerge from the spinal cord in the roots of the sixth thoracic to the first lumbar spinal nerve, and pass to the celiac plexus by way of the splanchnic nerves. Motor fibers may occur also in the vagi. Sensory fibers capable of causing a reflex constriction or dilation of the bladder are found in both the vagus and splanchnic nerves. Stimulation of the central end of the cut splanchnic causes a dilation of the bladder (reflex stimulation of the inhibitory fibers), which stimulation of the central end of the vagus causes a contraction of the bladder and a dilation (inhibition) of the sphincter muscle at the opening of the common duct into the intestine. These latter movements are the ones that occur during normal digestion, and we may assume, therefore, that in the normal reflex emptying of the gall-bladder the afferent path of the reflex is formed by the vagus fibers while the efferent path is through the splanchnic nerves."

Doyon's beautiful physiology can be confirmed on healthy dogs. It does not apply to men with diseased gall-bladders because the extensive destruction or infiltration of the neuro-muscular apparatus makes this beautiful reciprocal function difficult or impossible.

The first experiments on reciprocal or contrary innervation between the gall-bladder and the sphincter of Oddi is not the work of Meltzer. He merely confirmed the experiments previously made by Doyon. Reciprocal innervation is the work of Doyon. He claimed it for healthy dogs, not men.

## VISCERAL ADHESIONS AND BANDS: NORMAL INCIDENCE (SECOND PAPER)<sup>1</sup>

JOHN BRYANT

*Boston, Massachusetts*

At the last annual meeting of this Association, the writer presented the first of a series of papers on visceral adhesions, based upon a study of 297 unselected postmortem sections of all ages and both sexes. This paper was entitled "Visceral adhesions and bands, a preliminary report."<sup>2</sup> Its object was to indicate the scope, methods, and some of the conclusions arrived at as a result of the investigation being reported upon.

The object of the present communication is to make available in tabular form what may be called a standard of expectation of frequency of visceral involvement by adhesions in both sexes. This standard is given for all ages, and for four separate age periods. The tables themselves are comparable to and based upon the same original material as a table indicating the frequency of visceroptosis in unselected material at all ages, which was presented at the last annual meeting of the American Medical Association under the title "Visceroptosis: normal incidence, a preliminary report."<sup>3</sup>

There exists in the literature no series of cases with which the present can be compared. Therefore, although it is realized that greater accuracy might have been obtained from the study of a larger number of cases, the present results are offered in the belief that they will prove reasonably reliable, and in the hope that they may at least stimulate other workers to prove their accuracy or otherwise by reporting larger series of cases.

With regard to the tables themselves, it may be explained that the two main age groups "Below Forty" and "Above Forty" include the two other age subdivisions of "Fetal" and "Senile." Thus the age of

<sup>1</sup> Original data for this article obtained in 1912-1914 through the courtesy of Professors Pick and von Hanseman of Berlin, Professor Frankel of Hamburg and the students then working in their pathological institutes.

<sup>2</sup> Bryant: *Amer. Jour. Med. Sci.*, 1922, clxiii, 75.

<sup>3</sup> Bryant: *Jour. Amer. Med. Assoc.*, 1921, lxxvii, 1400.

forty becomes a proved dividing line between youth and age. The column entitled "No. of Obs." appearing in table I, might better have been entitled "Actual Frequency," since the figures in this column represent in each case the number of positive findings with reference to the total number of cases in each sex group. In table VI, the column "No. of Obs." indicates the total number of viscera involved by adhesions in the given age-sex group. In table VIII, the same column, "No. of Obs." indicates the total number of actual adhesions present in each age-sex group, the number of different types of adhesions that are present in any age-sex group being indicated in the main column of the table. In table VII, under "Legend," appear for the sake of brevity the expressions "2 to 8 adhesions present" and "8 or more adhesions present." It might have been more correct and more explicit to have used the phrases "2 to 8 viscera involved by adhesions" and "8 or more viscera involved by adhesions." With these exceptions, it is hoped that the tables will prove reasonably self-explanatory.

A glance at table I indicates that in the average case coming for examination, the transverse colon may be expected to be involved by adhesions more often than any of the other viscera; 7 women and 8 men out of every 10 presumably have some involvement of this structure by congenital or acquired adhesions. The gall-bladder and duodenum come next in order of frequency of involvement, these viscera being followed in frequency in the order named by the peritoneum, omentum, ascending colon, and hepatic flexure. The appendix, liver, and descending colon are also involved by adhesions sufficiently often to attract attention on a graphic chart. A study of table 1 also indicates that although the difference in the rate of involvement of the two sexes for any given organ seldom varies by as much as 10 per cent, neither sex has an excess of involvement of all the viscera studied. Thus in the first group of the three viscera most often involved by adhesions, the male percentage of involvement is higher than the female with regard to the transverse colon and gall-bladder, but lower than the female with regard to the duodenum. Also, the male percentage of involvement is greater as regards the omentum, ascending colon, appendix, and hepatic flexure, but lower for the peritoneum.

A study of tables II to V inclusive, allowing a consideration of progressive age changes in both sexes reveals, as would naturally be supposed, more facts of interest than can be obtained from a consideration of a single mixed-age table. Furthermore, a study of these tables is

sufficient to give a basis for inference concerning the congenital or acquired character of the adhesions found involving any individual viscus.

A careful preliminary study of all the available data, when arranged by successive age decades, caused the age of forty to be taken as a

| NO.<br>OF<br>OBS | VISCERA<br>INVOLVED | SEX            | PERCENTAGE FREQUENCY |    |              |              |                    |    |              |              |    |     |
|------------------|---------------------|----------------|----------------------|----|--------------|--------------|--------------------|----|--------------|--------------|----|-----|
|                  |                     |                | 10                   | 20 | 30           | 40           | 50                 | 60 | 70           | 80           | 90 | 100 |
| 62<br>33         | OMENTUM             | MALE<br>FEMALE |                      |    |              | 34.4<br>28.2 |                    |    |              |              |    |     |
| 67<br>46         | PERITONEUM          | MALE<br>FEMALE |                      |    |              | 37.2<br>39.3 |                    |    |              |              |    |     |
| 3<br>4           | RIGHT KIDNEY        | MALE<br>FEMALE | 1.7<br>3.4           |    |              |              |                    |    |              |              |    |     |
| 31<br>19         | LIVER               | MALE<br>FEMALE |                      |    | 17.2<br>16.2 |              |                    |    |              |              |    |     |
| 130<br>75        | GALL<br>BLADDER     | MALE<br>FEMALE |                      |    |              |              |                    |    |              | 72.2<br>64.1 |    |     |
| 101<br>74        | DUODENUM            | MALE<br>FEMALE |                      |    |              |              |                    |    | 56.1<br>63.2 |              |    |     |
| 1<br>2           | SMALL<br>INTESTINE  | MALE<br>FEMALE | 0.6<br>1.7           |    |              |              |                    |    |              |              |    |     |
| 5<br>6           | TERMINAL<br>ILEUM   | MALE<br>FEMALE | 2.8<br>5.1           |    |              |              |                    |    |              |              |    |     |
| 36<br>11         | APPENDIX            | MALE<br>FEMALE |                      |    | 20.0<br>9.4  |              |                    |    |              |              |    |     |
| 4<br>3           | CAECUM              | MALE<br>FEMALE | 2.2<br>2.6           |    |              |              |                    |    |              |              |    |     |
| 57<br>31         | ASCENDING<br>COLON  | MALE<br>FEMALE |                      |    |              | 31.7<br>26.5 |                    |    |              |              |    |     |
| 31<br>14         | HEPATIC<br>FLEXURE  | MALE<br>FEMALE |                      |    | 17.2<br>12.0 |              |                    |    |              |              |    |     |
| 144<br>82        | TRANSVERSE<br>COLON | MALE<br>FEMALE |                      |    |              |              |                    |    |              | 80.0<br>70.1 |    |     |
| 4<br>3           | SPLENIC<br>FLEXURE  | MALE<br>FEMALE | 2.2<br>2.6           |    |              |              |                    |    |              |              |    |     |
| 19<br>7          | DESCENDING<br>COLON | MALE<br>FEMALE |                      |    | 10.6<br>6.0  |              |                    |    |              |              |    |     |
| 13<br>2          | SIGMOID<br>FLEXURE  | MALE<br>FEMALE |                      |    | 7.2<br>1.7   |              |                    |    |              |              |    |     |
| 0<br>3           | RECTUM              | MALE<br>FEMALE | NONE<br>2.6          |    |              |              |                    |    |              |              |    |     |
| 0<br>19          | ADNEXA              | MALE<br>FEMALE | NONE<br>12.0         |    |              |              |                    |    |              |              |    |     |
| MALE -           |                     |                | TOTAL CASES          |    |              | 180          | TOTAL OBSERVATIONS |    |              | 708          |    |     |
| FEMALE -         |                     |                | " "                  |    |              | 117          | " "                |    |              | 429          | (  |     |
| BOTH SEXES       |                     |                | " "                  |    |              | 297          | " "                |    |              | 1137         |    |     |

TABLE I

*Visceral involvement by adhesions*

Percentage frequency in relation to sex and age  
All ages

definite point sharply dividing youth and age; this is from the point of view of adhesive processes within the abdomen. Having arrived at this point in the study, the material at hand was arranged as shown in the tables, in the two main age groups "Below Forty" and "Above

Forty," further indication of the relation of age to adhesions being gained by utilizing the two sub-groups, "Fetal" and "Senile." Obviously, in the point of total cases the fetal and senile groups are too small to be more than suggestive; but they are at least suggestive.

| VISCERA INVOLVED | SEX    | PERCENTAGE FREQUENCY |      |                       |    |      |    |    |      |      |       |
|------------------|--------|----------------------|------|-----------------------|----|------|----|----|------|------|-------|
|                  |        | 10                   | 20   | 30                    | 40 | 50   | 60 | 70 | 80   | 90   | 100   |
| OMENTUM          | MALE   | 5.6                  |      |                       |    |      |    |    |      |      |       |
|                  | FEMALE | 6.3                  |      |                       |    |      |    |    |      |      |       |
| PERITONEUM       | MALE   |                      | 16.7 |                       |    |      |    |    |      |      |       |
|                  | FEMALE |                      |      |                       |    | 43.8 |    |    |      |      |       |
| RIGHT KIDNEY     | MALE   | NONE                 |      |                       |    |      |    |    |      |      |       |
|                  | FEMALE | 6.3                  |      |                       |    |      |    |    |      |      |       |
| LIVER            | MALE   | 5.6                  |      |                       |    |      |    |    |      |      |       |
|                  | FEMALE | NONE                 |      |                       |    |      |    |    |      |      |       |
| GALL BLADDER     | MALE   |                      |      |                       |    |      |    |    |      |      |       |
|                  | FEMALE |                      |      |                       |    |      |    |    | 68.8 | 88.9 |       |
| DUODENUM         | MALE   |                      |      |                       |    |      |    |    |      |      |       |
|                  | FEMALE |                      |      |                       |    | 55.6 |    |    |      |      |       |
| SMALL INTESTINE  | MALE   | NONE                 |      |                       |    |      |    |    |      |      |       |
|                  | FEMALE | NONE                 |      |                       |    |      |    |    |      |      |       |
| TERMINAL ILEUM   | MALE   | 5.6                  |      |                       |    |      |    |    |      |      |       |
|                  | FEMALE |                      | 25.0 |                       |    |      |    |    |      |      |       |
| APPENDIX         | MALE   | 5.6                  |      |                       |    |      |    |    |      |      |       |
|                  | FEMALE |                      | 12.5 |                       |    |      |    |    |      |      |       |
| CAECUM           | MALE   | NONE                 |      |                       |    |      |    |    |      |      |       |
|                  | FEMALE | NONE                 |      |                       |    |      |    |    |      |      |       |
| ASCENDING COLON  | MALE   |                      | 11.1 |                       |    |      |    |    |      |      |       |
|                  | FEMALE | NONE                 |      |                       |    |      |    |    |      |      |       |
| HEPATIC FLEXURE  | MALE   |                      | 11.1 |                       |    |      |    |    |      |      |       |
|                  | FEMALE | 6.3                  |      |                       |    |      |    |    |      |      | 105.6 |
| TRANSVERSE COLON | MALE   |                      |      |                       |    |      |    |    |      |      |       |
|                  | FEMALE |                      |      |                       |    |      |    |    | 68.8 |      |       |
| SPLENIC FLEXURE  | MALE   | 5.6                  |      |                       |    |      |    |    |      |      |       |
|                  | FEMALE | NONE                 |      |                       |    |      |    |    |      |      |       |
| DESCENDING COLON | MALE   | 5.6                  |      |                       |    |      |    |    |      |      |       |
|                  | FEMALE | 6.3                  |      |                       |    |      |    |    |      |      |       |
| SIGMOID FLEXURE  | MALE   | NONE                 |      |                       |    |      |    |    |      |      |       |
|                  | FEMALE | NONE                 |      |                       |    |      |    |    |      |      |       |
| RECTUM           | MALE   | NONE                 |      |                       |    |      |    |    |      |      |       |
|                  | FEMALE | NONE                 |      |                       |    |      |    |    |      |      |       |
| ADNEXA           | MALE   | NONE                 |      |                       |    |      |    |    |      |      |       |
|                  | FEMALE | NONE                 |      |                       |    |      |    |    |      |      |       |
| MALE -           |        | TOTAL CASES 18       |      | TOTAL OBSERVATIONS 58 |    |      |    |    |      |      |       |
| FEMALE -         |        | " " 16               |      | " "                   |    | 47   |    |    |      |      |       |
| BOTH SEXES       |        | " " 34               |      | " "                   |    | 105  |    |    |      |      |       |

TABLE II

*Visceral involvement by adhesions*

Percentage frequency in relation to sex and age  
Fetal (12 to 55 cm.)

The most striking fact about table II, based upon a study of the fetal group, is that it should be possible to make any table worth showing at all, graphically, since it has for the most part long been considered that adhesions in the fetus are the exception rather than the rule. The second striking fact about this table, is that the percentages

should run so high for the viscera which are chiefly involved. It is for example rather startling to find 105 per cent of the transverse colons in this fetal group of 18 males involved by demonstrable adhesions, this statement being explained by the fact that many of the cases under consideration presented more than one definite band

| VISCERA INVOLVED | SEX    | PERCENTAGE FREQUENCY |      |      |      |                    |      |      |    |      |    |     |
|------------------|--------|----------------------|------|------|------|--------------------|------|------|----|------|----|-----|
|                  |        | 0                    | 10   | 20   | 30   | 40                 | 50   | 60   | 70 | 80   | 90 | 100 |
| OMENTUM          | MALE   |                      |      | 18.2 |      |                    |      |      |    |      |    |     |
|                  | FEMALE |                      | 11.5 |      |      |                    |      |      |    |      |    |     |
| PERITONEUM       | MALE   |                      |      |      | 30.7 |                    |      |      |    |      |    |     |
|                  | FEMALE |                      |      |      | 27.9 |                    |      |      |    |      |    |     |
| RIGHT KIDNEY     | MALE   | 1.1                  |      |      |      |                    |      |      |    |      |    |     |
|                  | FEMALE |                      | 6.6  |      |      |                    |      |      |    |      |    |     |
| LIVER            | MALE   |                      | 6.8  |      |      |                    |      |      |    |      |    |     |
|                  | FEMALE |                      |      | 13.1 |      |                    |      |      |    |      |    |     |
| GALL BLADDER     | MALE   |                      |      |      |      |                    |      |      |    | 69.3 |    |     |
|                  | FEMALE |                      |      |      |      |                    |      |      |    | 67.2 |    |     |
| DUODENUM         | MALE   |                      |      |      |      |                    | 52.3 |      |    |      |    |     |
|                  | FEMALE |                      |      |      |      |                    |      | 60.7 |    |      |    |     |
| SMALL INTESTINE  | MALE   | 1.1                  |      |      |      |                    |      |      |    |      |    |     |
|                  | FEMALE | 1.6                  |      |      |      |                    |      |      |    |      |    |     |
| TERMINAL ILEUM   | MALE   | 2.3                  |      |      |      |                    |      |      |    |      |    |     |
|                  | FEMALE |                      | 6.6  |      |      |                    |      |      |    |      |    |     |
| APPENDIX         | MALE   |                      |      | 18.2 |      |                    |      |      |    |      |    |     |
|                  | FEMALE |                      | 4.9  |      |      |                    |      |      |    |      |    |     |
| CAECUM           | MALE   | 2.3                  |      |      |      |                    |      |      |    |      |    |     |
|                  | FEMALE | NONE                 |      |      |      |                    |      |      |    |      |    |     |
| ASCENDING COLON  | MALE   |                      |      | 19.3 |      |                    |      |      |    |      |    |     |
|                  | FEMALE |                      |      | 16.4 |      |                    |      |      |    |      |    |     |
| HEPATIC FLEXURE  | MALE   |                      |      | 17.0 |      |                    |      |      |    |      |    |     |
|                  | FEMALE |                      | 11.5 |      |      |                    |      |      |    |      |    |     |
| TRANSVERSE COLON | MALE   |                      |      |      |      |                    |      |      |    | 70.4 |    |     |
|                  | FEMALE |                      |      |      |      |                    |      | 59.0 |    |      |    |     |
| SPLENIC FLEXURE  | MALE   | 2.3                  |      |      |      |                    |      |      |    |      |    |     |
|                  | FEMALE | NONE                 |      |      |      |                    |      |      |    |      |    |     |
| DESCENDING COLON | MALE   |                      | 5.7  |      |      |                    |      |      |    |      |    |     |
|                  | FEMALE |                      | 3.3  |      |      |                    |      |      |    |      |    |     |
| SIGMOID FLEXURE  | MALE   |                      | 5.7  |      |      |                    |      |      |    |      |    |     |
|                  | FEMALE | NONE                 |      |      |      |                    |      |      |    |      |    |     |
| RECTUM           | MALE   | NONE                 |      |      |      |                    |      |      |    |      |    |     |
|                  | FEMALE | 1.6                  |      |      |      |                    |      |      |    |      |    |     |
| ADNEXA           | MALE   | NONE                 |      |      |      |                    |      |      |    |      |    |     |
|                  | FEMALE | 4.9                  |      |      |      |                    |      |      |    |      |    |     |
| MALE             |        | TOTAL CASES          |      | 88   |      | TOTAL OBSERVATIONS |      | 284  |    |      |    |     |
| FEMALE           |        | "                    |      | 61   |      | "                  |      | 181  |    |      |    |     |
| BOTH SEXES       |        | "                    |      | 149  |      | "                  |      | 465  |    |      |    |     |

TABLE III  
*Visceral involvement by adhesions*

Percentage frequency in relation to age and sex  
Below forty years of age

involving the transverse colon. Incidentally, the method of recording the presence of these adhesions may be indicated by the following example: a fetus presents one adhesive band running from the gall-bladder across the duodenum to the transverse colon, and another

band running from the ascending to transverse colon; this case would be recorded as having a total of five viscera involved by adhesions, i.e., the gall-bladder, duodenum and ascending colon once each, and the transverse colon twice. As in table I, sex differences in the rate of involvement are conspicuous in table II. Thus whereas the trans-

| VISCERA INVOLVED | SEX    | PERCENTAGE FREQUENCY |    |      |                    |    |      |    |      |      |      |     |
|------------------|--------|----------------------|----|------|--------------------|----|------|----|------|------|------|-----|
|                  |        | 0                    | 10 | 20   | 30                 | 40 | 50   | 60 | 70   | 80   | 90   | 100 |
| OMENTUM          | MALE   |                      |    |      |                    |    | 50.0 |    |      |      |      |     |
|                  | FEMALE |                      |    |      |                    |    | 46.4 |    |      |      |      |     |
| PERITONEUM       | MALE   |                      |    |      |                    |    | 43.5 |    |      |      |      |     |
|                  | FEMALE |                      |    |      |                    |    | 51.8 |    |      |      |      |     |
| RIGHT KIDNEY     | MALE   | 2.2                  |    |      |                    |    |      |    |      |      |      |     |
|                  | FEMALE | NONE                 |    |      |                    |    |      |    |      |      |      |     |
| LIVER            | MALE   |                      |    |      | 27.2               |    |      |    |      |      |      |     |
|                  | FEMALE |                      |    | 19.6 |                    |    |      |    |      |      |      |     |
| GALL BLADDER     | MALE   |                      |    |      |                    |    |      |    |      | 75.0 |      |     |
|                  | FEMALE |                      |    |      |                    |    |      |    | 60.7 |      |      |     |
| DUODENUM         | MALE   |                      |    |      |                    |    |      |    | 59.8 |      |      |     |
|                  | FEMALE |                      |    |      |                    |    |      |    | 66.1 |      |      |     |
| SMALL INTESTINE  | MALE   | NONE                 |    |      |                    |    |      |    |      |      |      |     |
|                  | FEMALE | 1.8                  |    |      |                    |    |      |    |      |      |      |     |
| TERMINAL ILEUM   | MALE   | 3.3                  |    |      |                    |    |      |    |      |      |      |     |
|                  | FEMALE | 3.6                  |    |      |                    |    |      |    |      |      |      |     |
| APPENDIX         | MALE   |                      |    |      | 21.7               |    |      |    |      |      |      |     |
|                  | FEMALE |                      |    | 14.3 |                    |    |      |    |      |      |      |     |
| CAECUM           | MALE   | 2.2                  |    |      |                    |    |      |    |      |      |      |     |
|                  | FEMALE | 5.4                  |    |      |                    |    |      |    |      |      |      |     |
| ASCENDING COLON  | MALE   |                      |    |      |                    |    | 43.5 |    |      |      |      |     |
|                  | FEMALE |                      |    |      |                    |    | 37.5 |    |      |      |      |     |
| HEPATIC FLEXURE  | MALE   |                      |    | 17.4 |                    |    |      |    |      |      |      |     |
|                  | FEMALE |                      |    | 12.5 |                    |    |      |    |      |      |      |     |
| TRANSVERSE COLON | MALE   |                      |    |      |                    |    |      |    |      |      | 89.1 |     |
|                  | FEMALE |                      |    |      |                    |    |      |    |      | 82.1 |      |     |
| SPLENIC FLEXURE  | MALE   | 2.2                  |    |      |                    |    |      |    |      |      |      |     |
|                  | FEMALE | 5.4                  |    |      |                    |    |      |    |      |      |      |     |
| DESCENDING COLON | MALE   |                      |    |      | 15.2               |    |      |    |      |      |      |     |
|                  | FEMALE |                      |    | 8.9  |                    |    |      |    |      |      |      |     |
| SIGMOID FLEXURE  | MALE   |                      |    | 8.7  |                    |    |      |    |      |      |      |     |
|                  | FEMALE |                      |    | 3.6  |                    |    |      |    |      |      |      |     |
| RECTUM           | MALE   | NONE                 |    |      |                    |    |      |    |      |      |      |     |
|                  | FEMALE | 3.6                  |    |      |                    |    |      |    |      |      |      |     |
| ADNEXA           | MALE   | NONE                 |    |      |                    |    |      |    |      |      |      |     |
|                  | FEMALE |                      |    | 19.6 |                    |    |      |    |      |      |      |     |
| MALE             |        | TOTAL CASES          |    | 92   | TOTAL OBSERVATIONS |    | 424  |    |      |      |      |     |
| FEMALE           |        | "                    | "  | 56   | "                  |    | "    |    |      |      |      |     |
| BOTH SEXES       |        | "                    | "  | 148  | "                  |    | "    |    |      |      |      |     |

TABLE IV

*Visceral involvement by adhesions*

Percentage frequency in relation to sex and age  
Above forty years of age

verse colon, gall-bladder, and duodenum are involved more often in the male than in the female fetus, the peritoneum, terminal ileum, appendix and omentum are more frequently involved in the female than in the male fetus.

As one studies tables II to V with reference to the relationship of progressive age to visceral involvement by adhesions, several points of interest are brought to the attention. Among these points of interest may be mentioned the fact that the rate of involvement for any given viscus may either increase or decrease with age in both sexes. Also,

| VISCERA INVOLVED | SEX    | PERCENTAGE FREQUENCY |      |      |                    |      |      |      |    |       |        |
|------------------|--------|----------------------|------|------|--------------------|------|------|------|----|-------|--------|
|                  |        | 0                    | 10   | 20   | 30                 | 40   | 50   | 60   | 70 | 80    | 90 100 |
| OMENTUM          | MALE   |                      |      |      |                    |      |      |      |    | 64.7  |        |
|                  | FEMALE |                      |      |      |                    |      |      | 50.0 |    |       |        |
| PERITONEUM       | MALE   |                      |      |      |                    | 36.3 |      |      |    |       |        |
|                  | FEMALE |                      |      |      |                    |      |      | 55.6 |    |       |        |
| RIGHT KIDNEY     | MALE   | NONE                 |      |      |                    |      |      |      |    |       |        |
|                  | FEMALE | NONE                 |      |      |                    |      |      |      |    |       |        |
| LIVER            | MALE   |                      |      |      |                    | 29.4 |      |      |    |       |        |
|                  | FEMALE |                      |      |      |                    | 33.3 |      |      |    |       |        |
| GALL BLADDER     | MALE   |                      |      |      |                    |      |      |      |    | 76.5  |        |
|                  | FEMALE |                      |      |      |                    |      |      | 50.0 |    |       |        |
| DUODENUM         | MALE   |                      |      |      |                    |      |      | 47.1 |    |       |        |
|                  | FEMALE |                      |      |      |                    |      |      | 50.0 |    |       |        |
| SMALL INTESTINE  | MALE   | NONE                 |      |      |                    |      |      |      |    |       |        |
|                  | FEMALE |                      | 5.6  |      |                    |      |      |      |    |       |        |
| TERMINAL ILEUM   | MALE   | NONE                 |      |      |                    |      |      |      |    |       |        |
|                  | FEMALE | NONE                 |      |      |                    |      |      |      |    |       |        |
| APPENDIX         | MALE   |                      |      | 11.8 |                    |      |      |      |    |       |        |
|                  | FEMALE |                      |      | 11.1 |                    |      |      |      |    |       |        |
| CAECUM           | MALE   | NONE                 |      |      |                    |      |      |      |    |       |        |
|                  | FEMALE | NONE                 |      |      |                    |      |      |      |    |       |        |
| ASCENDING COLON  | MALE   |                      |      |      |                    |      | 35.3 |      |    |       |        |
|                  | FEMALE |                      |      |      |                    |      | 33.3 |      |    |       |        |
| HEPATIC FLEXURE  | MALE   |                      |      |      |                    |      | 29.4 |      |    |       |        |
|                  | FEMALE |                      |      | 11.1 |                    |      |      |      |    |       |        |
| TRANSVERSE COLON | MALE   |                      |      |      |                    |      |      |      |    | 88.2  |        |
|                  | FEMALE |                      |      |      |                    |      |      |      |    | 100.0 |        |
| SPLENIC FLEXURE  | MALE   | NONE                 |      |      |                    |      |      |      |    |       |        |
|                  | FEMALE |                      | 11.1 |      |                    |      |      |      |    |       |        |
| DESCENDING COLON | MALE   |                      |      |      |                    |      | 35.3 |      |    |       |        |
|                  | FEMALE |                      |      |      |                    | 16.7 |      |      |    |       |        |
| SIGMOID FLEXURE  | MALE   |                      |      |      |                    |      | 23.5 |      |    |       |        |
|                  | FEMALE |                      |      | 11.1 |                    |      |      |      |    |       |        |
| RECTUM           | MALE   | NONE                 |      |      |                    |      |      |      |    |       |        |
|                  | FEMALE | NONE                 |      |      |                    |      |      |      |    |       |        |
| ADNEXA           | MALE   | NONE                 |      |      |                    |      |      |      |    |       |        |
|                  | FEMALE |                      |      |      |                    | 22.0 |      |      |    |       |        |
| MALE             |        | TOTAL CASES          |      | 17   | TOTAL OBSERVATIONS |      | 81   |      |    |       |        |
| FEMALE           |        | "                    |      | 18   | "                  |      | 83   |      |    |       |        |
| BOTH SEXES       |        | "                    |      | 35   | "                  |      | 164  |      |    |       |        |

TABLE V

*Visceral involvement by adhesions*

Percentage frequency in relation to sex and age  
Senile (over seventy years)

the relative frequency of involvement of any special viscus with regard to sex may vary at different periods. One or two examples may be given.

The terminal ileum shows in both sexes a decreasing involvement by adhesions with progressive age, the rate being distinctly highest in the

fetus. Incidentally, this finding is diametrically opposed to the dictum of Lane; he has stated that bands about the terminal ileum represent crystallization of lines of force which increase with age, this crystallization resulting from efforts of the organism to support a sagging viscus. The omentum in both sexes is increasingly involved with progressive age; the same progressive increase with age is evident as regards the adnexa of the female, and the sigmoid flexure in both sexes.

The sigmoid flexure is a conspicuous example of a viscus progressively involved with increasing age. No involvement occurs in the fetus in either sex. Below the age of forty in the male there is an involvement of but 5.7 per cent. The rate increases to 8.7 per cent in the male above forty, and the figure for the senile male rises sharply

| NO. OF CASES | NO. OF OBS. | AGE      | SEX    | AVERAGE NUMBER OF VISCERA INVOLVED |   |     |   |   |    | INCREASE OVER FOETAL RATE PERCENTAGE |    |      |    |
|--------------|-------------|----------|--------|------------------------------------|---|-----|---|---|----|--------------------------------------|----|------|----|
|              |             |          |        | 0                                  | 2 | 4   | 6 | 8 | 10 | 20                                   | 40 | 60   | 80 |
| 18           | 58          | FOETAL   | MALE   |                                    |   | 3.2 |   |   |    |                                      |    |      |    |
| 16           | 47          |          | FEMALE |                                    |   | 2.9 |   |   |    |                                      |    |      |    |
| 88           | 284         | BELOW 40 | MALE   |                                    |   | 3.2 |   |   |    | NONE                                 |    |      |    |
| 61           | 181         |          | FEMALE |                                    |   | 3.0 |   |   |    | 3.4                                  |    |      |    |
| 92           | 424         | ABOVE 40 | MALE   |                                    |   | 4.6 |   |   |    |                                      |    | 43.8 |    |
| 56           | 248         |          | FEMALE |                                    |   | 4.4 |   |   |    |                                      |    | 51.7 |    |
| 17           | 81          | SENILE   | MALE   |                                    |   | 4.8 |   |   |    |                                      |    | 50.0 |    |
| 18           | 83          |          | FEMALE |                                    |   | 4.6 |   |   |    |                                      |    | 58.7 |    |

TABLE VI

*Average number of viscera involved by adhesions*

*In relation to age and sex*

to 23.5 per cent of involvement. The figures for the female with regard to increased age involvement are less striking but the same in character.

The female adnexa present another striking example of what one must look upon as adhesive processes of degeneration. Here, the involvement is but 4.9 per cent below the age of forty, as contrasted with a rate of 19.6 per cent for the female group above forty years of age, and a rate of 22 per cent for the female senile group.

The peritoneum is an example of a viscus varying with age in its relative frequency as to sex involvement. Thus, although the rate is much higher in the female than the male fetus, the figures are reversed in the group below forty years of age: the figures are however reversed again in the group above forty years of age; furthermore, the peritoneum becomes increasingly more involved in the senile female, but less involved in the senile male group.

It is believed that even the few examples given above are sufficient to prove that in any study of the subject of visceral adhesions and bands,

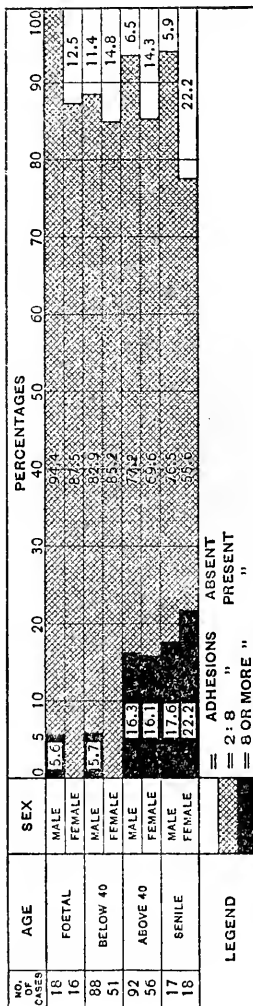


TABLE VII  
*Variation in complexity of adhesions present*  
 In relation to age and sex

both age and sex factors must be carefully considered before any reliable results can be arrived at. In general, the problem of visceral adhesions may be approached from at least two main points of view. The first is that of the involvement of the individual viscera by adhesive processes either congenital or acquired; this is the point of view from which tables I to V inclusive have been considered.

The second point of view from which the problem of visceral adhesions may be approached, is that of the actual adhesions themselves. For example, the congenital band frequently found running from the gall-bladder down across the duodenum to the transverse colon, if studied from the first point of view, would be considered under the

| NO. OF CASES | NO. OF OBS. | AGE          | SEX    | ACTUAL NUMBER OF DIFFERENT ADHESIONS PRESENT |   |    |    |    |    |
|--------------|-------------|--------------|--------|--|---|----|----|----|----|
|              |             |              |        | 0  | 5 | 10 | 15 | 20 | 25 |
| 18           | 58          | FOETAL       | MALE   |  |   | 10 |    |    |    |
| 16           | 47          |              | FEMALE |  | 8 |    |    |    |    |
| 10           | 26          | BIRTH-2 YRS. | MALE   |  | 8 |    |    |    |    |
| 8            | 16          |              | FEMALE | 5  |   |    |    |    |    |
| 12           | 30          | 2-10         | MALE   |  |   | 10 |    |    |    |
| 11           | 30          |              | FEMALE |  |   | 9  |    |    |    |
| 11           | 36          | 10-20        | MALE   |  |   | 10 |    |    |    |
| 1            | 2           |              | FEMALE | 1  |   |    |    |    |    |
| 13           | 48          | 20-30        | MALE   |  |   | 11 |    |    |    |
| 11           | 38          |              | FEMALE |  |   | 11 |    |    |    |
| 24           | 86          | 30-40        | MALE   |  |   |    |    | 20 |    |
| 14           | 48          |              | FEMALE |  |   |    | 15 |    |    |
| 23           | 104         | 40-50        | MALE   |  |   |    |    |    | 23 |
| 16           | 77          |              | FEMALE |  |   |    | 18 |    |    |
| 31           | 142         | 50-60        | MALE   |  |   |    |    |    | 23 |
| 15           | 59          |              | FEMALE |  |   |    | 14 |    |    |
| 21           | 97          | 60-70        | MALE   |  |   |    |    | 20 |    |
| 7            | 29          |              | FEMALE |  | 8 |    |    |    |    |
| 17           | 81          | 70-80        | MALE   |  |   |    |    | 19 |    |
| 18           | 83          |              | FEMALE |  |   |    |    |    | 25 |

TABLE VIII

*Total number of variations in types of adhesions present*

*In relation to age and sex*

three separate headings of gall-bladder, duodenum, and transverse colon. On the other hand, if studied from the second point of view of the adhesive process itself, this congenital band would be considered as a single distinct structure. It is from this second point of view that tables VI, VII, and VIII approach the subject of visceral adhesions, from three particular angles.

The object of table VI is to indicate the surprising increase in the average number of viscera involved by adhesions with increasing age, in both sexes. It will be seen that below the age of forty the average number of viscera involved does not exceed four per case; whereas, above the age of forty the average rate of involvement is nearer five

than four per case. Reducing this question to percentage increase above the fetal rate of involvement, it is shown that whereas there is no percentage increase below the age of forty, the increase after this age averages about 50 per cent for both sexes. It is difficult to explain this striking 50 per cent increase on any other basis than that of some increasing degenerative tendency to adhesions, possibly based upon decreased resistance to the irritative and infective trauma to which all viscera are exposed in varying and progressive degrees at all ages in both sexes.

The relation of age to the complexity of adhesions, or the number of viscera involved in any single adhesive process is clearly shown in table VII. From this one learns that the adhesions found in the fetus are relatively uncomplicated in character, and that complexity is practically a synonym for age. In other words, the increasing complexity of adhesions with age seems to be an indication of the decreasing resistance of the viscera to the trauma of all kinds to which they are subjected throughout life.

It has been shown in tables VI and VII that the age of forty is critical in relation to the average number of viscera involved, and in relation to the complexity of the adhesions themselves. A study of table VIII leads to the conclusion that the total variety of adhesions present in any age group also increases with age, but that the marked increase occurs in this case in the thirty-forty-year decade, or ten years before the onset of the two other age changes just referred to.

In summarizing these various age changes, it may be said that the distinguishing characteristics of such congenital or developmental adhesions as are found in the fetus are, first, the limited average number of viscera involved, and second, the simplicity and absence of variety in type of the adhesions themselves. The acquired and degenerative adhesions of age, are on the contrary characterized by the large average number of viscera involved in any given process, the great increase in the variety of adhesions found, and the strikingly increased complexity of the adhesions themselves.

#### CONCLUSIONS

1. The transverse colon is more frequently involved by adhesions than any other abdominal viscus. Seven women and 8 men out of every 10 persons presumably have some involvement of this viscus by congenital or acquired adhesions. Next in frequency of involvement come, in the order named, the gall-bladder, duodenum, peri-

toneum, omentum, ascending colon, hepatic flexure, appendix, liver and descending colon.

2. Within a range of variation not usually exceeding 10 per cent, the rate of involvement of a given viscus may be greater in the male or in the female, and this relative rate of involvement with regard to sex may further vary with age.

3. The rate of involvement by adhesions, is for several viscera higher in the fetus than at later ages, as for the transverse colon in the male and the terminal ileum in the female.

4. The rate of involvement by adhesions increases rapidly with progressive age for certain other viscera, as for the sigmoid flexure in the male and the adnexa of the female.

5. The age of forty is critical in both sexes with reference to the average number of viscera involved by adhesions in any given case. After the age of forty, there is a sudden increase of involvement by about 50 per cent in both sexes, the increase being somewhat more marked in the male than in the female.

6. Complexity is practically a synonym for age, with regard to the number of viscera involved in any given adhesive process. This increase in complexity amounts to over 200 per cent after forty years of age.

7. Variety in the character of the adhesions present also increases with age. A sudden marked increase of nearly 100 per cent occurs in the thirty-forty-year decade, or ten years earlier than the onset of the marked increase with regard to the average number of viscera involved, and the onset of the marked increase in the complexity of the adhesions themselves.

8. The distinguishing characteristics of congenital or developmental adhesions, are simplicity and lack of variety in type.

9. The distinguishing characteristics of acquired adhesions, are complexity and variety in type.









